ROLE OF BACTERIAL VAGINOSIS IN PRETERM LABOUR

DISSERTATION SUBMITTED FOR

M.D (BRANCH – II)

(OBSTETRICS & GYNAECOLOGY)

MARCH 2010

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"ROLE OF BACTERIAL VAGINOSIS IN PRETERM LABOUR"** is a bonafide record work done by **Dr. N. PUNITHAMANI** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for M.D Branch II – Obstetrics & Gynaecology.

Dr. DILSHATH. M.D. D.G.O..
HOD & Professor
Department of O&G
Madurai Medical College,
Madurai.

DECLARATION

I **Dr. N. PUNITHA MANI** solemnly declare that the dissertation titled **"ROLE OF BACTERIAL VAGINOSIS IN PRETERM LABOUR"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of M.D degree Branch – II (Obstetrics & Gynecology) to be held in March 2010.

Place : Madurai **Dr. N. PUNITHA MANI**

Date :

ACKNOWLEDGEMENT

My sincere and thankful gratitude to **Prof. Dr. DILSHATH, M.D., D.G.O.,** Professor and Head of the Department of Obstetrics and Gynaecology who was of great help, from the beginning of the study and for her expert guidance throughout this study.

I wish to express my sincere thanks to my guide and teacher, Addl professor **Dr.S. Lalitha** for her valuable suggestion, support and guidance at every stage of this study.

I am very grateful to **Dr. Parvathavarthini, M.D., D.G.O., Dr. Subbulakshmi, M.D., D.G.O., Dr. Ankayarkanni M.D., D.C.H., Dr. Ambigai Meena, M.D., D.G.O.,** Additional Professors, Department of Obstetrics and Gynaecology, for their valuable suggestions in preparing this dissertation.

My grateful thanks to **Asst. Professors** of Department of Obstetrics and Gynaecology, for their help during this study.

I am extremely thankful to **THE DEAN**, Madurai Medical College and Government Rajaji Hospital, Madurai for granting me permission to undertake this study.

I wish to thank Prof. Dr. P.A.T. Jegadeeswari, M.D. Head of the Department, Microbiology, Madurai Medical College, for her immense help in my study.

Thanks to my fellow post graduates who had assisted me throughout the study.

Last but not the least, I am immensely grateful to all the patients who took part in this study.

CONTENTS

INTRODUCTION

Preterm labour with subsequent delivery of the premature baby is the major cause of perinatal morbidity and mortality.

Preterm birth is defined as birth before 37 week of gestation. Despite major advances in obstetric and neonatal care over the past decade the incidence remained constant at 10-15%.

Preterm premature rupture of membranes and spontaneous preterm labour account for approximately 80% of preterm deliveries. The remaining 20% are indicated deliveries for maternal or fetal reasons.

Preterm deliveries poses a problem because of the severe neonatal complications that often occur afterwards that includes death, respiratory distress syndrome, sepsis and necrotizing enterocolitis

The etiology of preterm labor (PTL) is multifactorial with increasing evidence that infection is a possible cause in upto 40% of cases. PTL may either be a physiological process occurring too early in pregnancy or a pathological process as a result of an abnormal signal such as infection.

Bacterial vaginosis and prediction of preterm Birth:

Normal genital tract flora are dominated by Lactobacillus which produce lactic acid keeping the vaginal pH below 4.5 so discouraging the growth of other organisms. During pregnancy the concentration of Lactobacillus species increase 10 fold as pregnancy progresses. Increased levels of lactobacilli make the vaginal ecosystem inhibitory to the growth of many pathogens. In Bacterial vaginosis lactobacilli are altered resulting in 1000 fold increase in anaerobes mobiluncus species and the genital mycoplasmas.

Bacterial vaginosis has been associated with preterm labour, PROM and with postpartum maternal and neonatal infections. Bacterial vaginosis has been strongly associated with vaginal cuff infections following hysterectomy, PID, post abortal PID and caesarean endometritis.

Vaginitis versus bacterial vaginosis

Vaginal infections such as Trichomonas vaginalis and candida generally induce inflammatory response in vaginal wall which is usually accompanied by increase number of leukocytes in vaginal fluid. This is the hallmark of "itis" condition.

The term bacterial vaginosis was introduced to describe increased vaginal discharge without signs of clinical inflammation and noticeable absence of leucocyte. The vaginosis was called bacterial because of absence of fungi and parasites as cause of syndrome.

AIM OF THE STUDY

- To findout the prevalence of bacterial vaginosis in spontaneous preterm labour.
- To study the association of bacterial vaginosis with preterm labour in a tertiary care institute.
- To determine whether the presence of bacterial vaginosis is significantly associated with the maternal and neonatal outcome.

REVIEW OF LITERATURE

History

Bacterial vaginosis is an alteration of the vaginal flora where normally predominant lactobacilli are replaced by cocktail of organisms including Gardnerella vaginalis and anaerobes. A review of the history of bacterial vaginosis allows historical perspective and provides a better understanding of this disease for the future.

The term “bacterial vaginosis” the currently accepted name for this disease has evolved over 100 years. An extensive study of vaginal flora was first described by Doderlein in 1892 where lactobacillus was first identified as the predominant organism found in normal vaginal flora. In Doderlein study the normal vaginal flora was considered to be homogenous, consisting of only gram-positive bacilli. Heterogenous flora was deemed unhealthy and the term non specific vaginitis was coined to describe this condition for the next 60 years.

In their landmark article in 1955 Gardner and Dukes described the clinical features and identified the organism thought to be responsible for the disease now known as bacterial vaginosis. Gardner and Dukes proposed that non specific vaginitis was in fact a specific entity caused by a sole organism Haemophilus vaginalis.

Thereafter the name of this syndrome became Haemophilus vaginalis vaginitis.

In 1963 the organism was subsequently termed corynebacterium vaginalis because of physiochemical differences from the Haemophilus species. In 1980, in honor of Dr.Herman Gardner the name of the organism was changed to Gardnerella vaginalis and the disease became known as Gardnerella vaginalis vaginitis.

Within the last decade more sophisticated culturing techniques became available. It became evident that Gardnerella vaginalis could be found in greater than 50% of women without signs and symptoms of vaginitis. In addition bacteria other than Gardnerella vaginalis were associated with bacterial vaginosis. The early 1980s saw an emergence of evidence that anaerobic bacteriae were responsible for the characteristic fishy odor of this disease and the term anaerobic vaginosis was coined by Blakwell and associates in 1982. In 1984, the term bacterial vaginosis was advocated to reflect the complex alteration of vaginal bacterial flora and to constitute the presence of increased discharge without an apparent inflammatory response.

Epidemiology and Risk Factors

These are several proposed risk factors for BV, some of which are still disputed. The trigger for the change from Lactobacillus – dominated flora to bacterial vaginosis associated flora has been linked to many possible factors including age at first sexual intercourses, change in sexual partners, greater number of life time sexual partners, and concurrent sexually transmitted disease. Cigarette smoking and the use of an intra-uterine contraceptive device are both linked to an increased risk of acquiring bacterial vaginosis. Vaginal douching has also been implicated as a risk factor for bacterial vaginosis, by aiding the ascent of microorganisms into the upper genital tract. There is also an evidence of racial disparity of bacterial vaginosis, which is seen to occur more frequently in women of Afro-Caribbean origin compared to Caucasian women.

Prevalence Study of Bacterial Vaginosis

1. **Obstetric patients**

In 1957, Gardner et al reported 10.1% of bacterial vaginosis in 1041 patient. Similar studies by many authors reported 10 to 31% of bacterial vaginosis. In India a study by P.Balamba from Hyderabad reported bacterial vaginosis in 35% of their cases.

2. **College students**

In a random group study Spiegel et al diagnosed bacterial vaginosis in 23%. Of those with bacterial vaginosis 97% were asymptomatic.

With the application of various criteria the prevalence of bacterial vaginosis ranged from 12% to 25% with an average of 20% in patients group studied and about 50% of those with bacterial vaginosis were asymptomatic.

3. Gynecological patients

In the first study by Gardner and Dukes of 579 gynaecologic patients prevalence rate was 13.3% In the study by Thomason on 500 non-pregnant patients, 70% had some form of vaginitis and 23%

had bacterial vaginosis. Similar study by various authors gives the prevalence rate between 10% and 62%.

4. Commercial sex workers

Bell et al diagnosed bacterial vaginosis in 23% of 35 commercial sex wokers in one study 26% of 57 commercial sex workers in another study. The age range of these patients was between 12 and 18 years.

5. Prevalence in STD clinics

Hill et al., diagnosed bacterial vaginosis in 37% of STD patients. Embree et al., diagnosed bacterial vaginosis in 20 of 33 patients which ranges to 61% . In 1975 the study from dentures for disease control reported bacterial vaginosis in 12.3% of 11,264 patients. So the range is highly variable.

Aetiology and predisposing factors

Even after many years of research, the underlying pathogenesis of bacterial vaginosis is still unknown. However it is clear that there is a strong association between Gardnarella vaginalis, anaerobic bacteria, Mycoplasma hominis and bacterial vaginosis. Patients with bacterial vaginosis have a distinct change in the vaginal flora resulting in a loss of lactobacilli, an increase in other flora and an elevated vaginal pH. Hydrogen peroxide producing strains of lactobacilli are usually found in women with normal vaginal flora but in only 6% of women with bacterial vaginosis.

Changes in microbial flora were found to occur more often during the follicular phase of the cycle at a time when oestrogen concentration was relatively high compared to progesterone. Studies show that the administration of oestrogen result in increased susceptibility to infection by Mycoplasma hominis and Neisseria gonorrhea and that there was a large increase in the number of organisms in the genital tract. This change was accompanied by the appearance of vaginal epithelial cells with many adherent organisms, described as ‘clue’ cells. The prevalence of bacterial vaginosis decreases as pregnancy progresses and, although the concentrations of oestrogens is elevated throughout, the relative concentrations of

oestrogens and progesterone alter as pregnancy progresses. If an endocrine change is the cause of bacterial vaginosis, the mechanism is unclear. One theory suggests that a change in endocrine status encourages the growth of endogenous bacteria, normally present in small numbers, or it may be that this change favours disappearance of Lactobacilli which allows unopposed growth of other organisms.

Other theories propose an enzymatic role in the pathogenesis of bacterial vaginosis. Mucinase and sialidase levels measured in samples of vaginal fluid in women with bacterial vaginosis were found to be significantly elevated compared to women with normal vaginal flora and it may be possible that they allow the entry of pathogens by promoting the breakdown of the mucosal barrier.

More recently, there has been evidence to suggest a role for phage viruses in the aetiology of bacterial vaginosis. A phage or bacteriophage is a virus that infects bacteria. They are capable of lysing bacteria and releasing further phages into the environment, or they can co-exist within the bacteria as parasites and exert their effect. It has been proven that phages can be isolated from vaginal Lactobacilli and invitro experiments show that these phages have the potential to infect vaginal Lactobacilli of other women.

BACTERIOLOGY

Normal Vaginal Flora

Resident vaginal flora consists of a combination of both aerobic and anaerobic organisms. The microflora of normal vaginal secretions is characterized by a predominance of lactobacilli primarily acidophilic lactobacilli. Usually an additional 5 to 15 bacterial species are also normally cultured from the vagina. Gardnerella vaginalis can be found in greater than 50% normal healthy women.

The vaginal flora can be divided into aerobic and anaerobic organisms. Common aerobic facultative organisms found include lactobacilli, staphylococcus epidermidis, streptococci and Gardnerella vaginalis. The anaerobic organisms commonly found include Bacteroides species, B.bivius and Peptostreptococcus.

Mycoplasma hominis can be found in 20% to 50% and ureaplasma urealyticum can be found in 50% to 70% of sexually active women. In women with normal vaginal flora lactobacillus species account for greater that 95% of the total organisms present.

In pregnancy, there is a rise in the overall numbers of vaginal flora compared to the non-pregnant state due mainly to an increase

in Lactobacilli by approximately 10-fold. There is a concurrent reduction in anaerobes but as relative stability of aerobes. With increasing gestation, the flora tend to become more benign, mainly due to the increasing numbers of Lactobacilli such that, at term, the vaginal flora is dominated by organism of low virulence which pose no threat to the fetus. Any alteration in this balance such as occurs in bacterial vaginosis, can result in adverse sequelae.

Bacteriology of bacterial vaginosis

Bacterial vaginosis is believed to represent a synergistic polymicrobial infection, characterized by an overgrowth of bacterial species normally found in the vagina. The lactobacilli dominated flora is replaced with a mixed predominantly an aerobic flora consisting of Gardnerella vaginalis, anaerobes such as bacteroides peptostreptococcus and Mycoplasma hominis.

PRIMARY PATHOGENS OF

BACTERIAL VAGINOSIS

Anaerobes	**Facultative Anaerobes**
Bacteriods	Gardnerella vaginalis
Peptostretoccus and Mobiluncus species	Mycoplasms hominis

Lactobacilli are absent or greatly reduced in most bacterial vaginosis patients. The concentration of bacteria increase 100-1000 fold in women with bacterial vaginosis compared to normal healthy women. Ureaplasma urealyticum is usually found about as often in women with bacterial vaginosis as in control groups and thus it is not thought to play a significant role in the disease.

Mycoplasma hominis is isolated more frequently from women with bacterial vaginosis than from normal healthy women. In one study, Gardnerella vaginalis, Mycoplasma hominis and anaerobes were recovered significantly more often in bacterial vaginosis patients than controls. The total bacterial count in the normal vaginal ecosystem is 10^5 to 10^6/ml ($<10^6$ per ml) secretions, but in bacterial vaginosis the concentrations increase greatly often to 10^9 to 10^{11}/ ml of secretions . Lactobacilli are characteristically absent or present only at very low concentrations in women with bacterial vaginosis. Only 6% of women with bacterial vaginosis have facultative H_2O_2 producing lactobacilli present compared to 96% of women with normal flora. This suggests that H_2O_2 producing lactobacilli may inhibit bacterial vaginosis by inhibiting overgrowth of Gardnerella and anaerobes.

Mobiluncus species have been detected by Gram stained smear or culture in approximately 40 – 60% of women with bacterial vaginosis. Mobiluncus are curved, gram variable flagellated, anaerobic, slow growing organism with a cork screw motility on a wet mount of vaginal fluid and in pure culture. Discovering Mobiluncus on a wet mount is an excellent indicator of bacterial vaginosis (positive predictive value 98.6%.)

G.vaginalis is a facultative aerobic, non-spore forming, non capsulated, non-motile, pleomorphic gram variable rod. G vaginalis is uniformly found in high percentage in upto 95% patients with bacterial vaginosis.

Role of vaginal Lactobacilli

Vaginal lactobacilli are widely assumed to protect against infection by genital pathogens. Production of estrogen by women of reproductive age increases glycogen content of the vaginal epithelium. Glycogen is then metabolized into glucose and subsequently to lactic acid mainly by lactobacilli. The presence of the lactic acid is responsible for the low pH in the vagina and the low pH favours growth of acidophilic organisms, such as lactobacilli.

Lactic acid production by Lactobacilli results in an acidic pH (3.8 to 4.2) in normal women and inhibits the growth of Gardnerella vaginalis and anaerobes(including mobiluncus species). In addition to lactic acid Lactobacilli produce a number of antibacterial compounds including acidolin, lactacin B and H_2O_2. H_2O_2 together with peroxidase and a halide ion appear capable of killing many catalase negative vaginal bacteria.

The pathogenesis of bacterial vaginosis is thought to include the elimination or reduction of antibacterial activity expressed by endogenous vaginal lactobacilli. Several mechanism of protection by Lactobacilli against infection have been proposed including.

a. Maintaining a low vaginal pH via lactic acid production.
b. Interference with the adherence of bacteria to epithelial cells.
c. Production of H_2O_2 (Bacterial antagonism).
d. Production of bacteriocins.

Role of vaginal pH

Normal vaginal pH is 3.8 to 4.2. The acidic environment of the normal vaginal microflora limits the growth of potentially pathogenic bacteria and protozoa.

In contrast bacterial vaginosis is characterized by an elevated pH (>4.5). Elevated vaginal pH is associated with loss of lactobacilli particularly H_2O_2 producing lactobacilli. Increased pH levels are generally associated with an increased ability of bacterial binding to eukaryotic cells. Adherence of Gardnerella vaginalis to human vaginal epithelial cells was high at a pH> 5.0 but limited at pH 3 to 4. Optimum growth of Mobiluncus species occurs at a pH greater than 5.

The adherence of Candida albicans to vaginal cells at pH 6 was considerably greater than at pH 3 to 4, a range that closely corresponds to the normal vaginal pH. Additionally when epithelial cells were coated with lactobacilli, there was significantly decreased adherence of candida albicans.

The absence of lactic acid and the production of succinate, which also raises vaginal pH, has been postulated to blunt the chemotactic response of polymorphonuclear leukocytes and to reduce their killing ability. This may explain why bacterial vaginosis produces no cellular inflammatory response despite the presence of high numbers of potentially pathogenic micro-organisms.

A variety of factors influence vaginal pH. Desquamation or trauma can alter the vaginal pH and hormonal influences can have the same effect.

Seminal fluid is basic (pH>7) and this causes a transient increase in vaginal pH following intercourse. The increase in pH cause the release of volatile amines and produces the characteristic fishy odor noted in bacterial vaginosis.

SIGNS AND SYMPTOMS

a. Bacterial vaginosis carry a variety of symptoms or none at all.

b. Greater than 50% are asymptomatic. The major symptoms of bacterial vaginosis present in 50% of cases is a malodorous vaginal discharge usually described as fishy or musty. The odor is caused by the alkaline volatilization of various amine by products of anaerobic metabolism. Exacerbation of the odor may occur following sexual intercourse or during menstruation as a result of a rise in vaginal pH.

c. Another common patient's complaint is an increased vaginal discharge. The patient may complain that the discharge stains her clothing. This discharge is usually thin, gray or white and homogenous and tends to adhere to the vaginal wall.

In contrast normal vaginal fluid is more viscous with a floccular consistency and tends to pool in the dependent recesses of the vagina. The common characteristics of vaginal discharge in healthy women and women with bacterial vaginosis are summarized in Table.

CHARACTERISTICS OF VAGINAL DISCHARGE

	Normal women	Women with bacterial vaginosis
Discharge appearance	White flocculent	Homogenous, gray, white
Odor	Odorless	Malodorous (fishy / musty)
pH	<4.5(3.8 to 4.2)	>4.5
Clue cells	Absent	Present
Lactobacilli	Predominant>95%	Absent or present in very low numbers
Other bacteriae	Gardnerella vaginalis at low concentration (in about 50% of women)	Mycoplasma hominis, anaerobes (Bacteroides, Mobiluncus) predominate

d. Vulval pruritis and irritation are not common features of bacterial vaginosis. Bacterial vaginosis is not an inflammatory condition. Polymorphonuclear leukocytes are not usually present in large quantities despite the presence of infection.

DIAGNOSIS

Composite clinical criteria

In 1983, Amsel developed a set of composite clinical criteria which are still widely used both in clinical practice and in research. The diagnosis is made by finding three of the following four signs:

I a homogenous vaginal discharge;

II an elevated vaginal pH> 4.5:

III a positive whiff test on addition of a solution of 10% potassium hydroxide (KOH) to a sample of vaginal secretions;

IV the presence of clue cells on microscopic examination of a wet preparation of vaginal secretions.

The presence of atleast three out of four of these criteria is regarded as diagnostic of bacterial vaginosis.

Homogenous vaginal discharge

The assessment of vaginal discharge is the most subjective of these, but still correlates better with the presence of bacterial vaginosis. However, it is important to realize that the absence of discharge does not imply the absence of bacterial vaginosis. It is not

accepted as a reliable indicator on its own as it is neither sensitive not specific to bacterial vaginosis.

Patients with bacterial vaginosis have a thin, copious, molodorous watery vaginal discharge that does not form clumps, is often present at the introitus and sticks to the anterior and lateral vaginal walls. This discharge can be distinguished from the normal vaginal discharge, which has a thick, milky, clumpy, appearance. The vaginal walls in patients with bacterial vaginosis appear normal and are not erythematous (suggesting candidiasis) and do not have a strawberry appearance (suggesting trichomoniasis). A yellow purulent or foamy discharge suggests cervicitis or other form of vaginitis.

pH

Vaginal pH is measured using narrow-range pH paper and assessing the colour change produced by a sample of vaginal secretion taken from the posterior fornix. A low pH virtually excludes bacterial vaginosis An elevated pH is the most sensitive, but least specific of the criteria used for the diagnosis of bacterial vaginosis, as an increase can also be associated with menstruation, recent sexual intercourse or infection with T.vaginalis.

Vaginal fluid pH determination is simple to perform and it is economical and has a high negative predictive value. Virtually no patient with bacterial vaginosis has a normal vaginal pH.

ODOUR

The whiff test involves the addition of a drop of 10% KOH to a sample of vaginal secretions which produces a characteristic fishy odour in the presence of bacterial vaginosis.

Subjective complaints of vaginal odor can be associated significantly with bacterial vaginosis. The odor often is described by the patients as "fishy". Pheifer et at., were the first to report the presence of such a characteristic odor. The odor may be recognized on speculum examination, but the intensity will increase with the addition of potassium hydroxide. The pH increase liberates certain amines, predominantly putrescine and cadaverine which are the decarboxylation products of arginine and lysine metabolism, respectively.

These compounds are non volatile salts but become volatile at alkaline pH and emit the fishy odour. Because semen has a pH of approximately 7 that, when ejaculated into the vagina, could increase

the pH: Therefore, the patient and her partner might notice a disagreeable fishy odor after intercourse.

As a single entity, the whiff test has a positive predictive value of 90% and a specificity of 70%.

Clue Cells

'Clue cells' are desquamated vaginal epithelial cells that are densely coated with adherent bacteria such that their borders are indistinct. The detection of clue cells on direct microscopy is the single most sensitive and specific criterion for bacterial vaginosis but it is operator – dependent. Debris and degenerated cells may be mistaken for clue cells and Lactobacilli may adhere to epithelial cells in low numbers. Clue cells can be identified on a Gram stain or a wet preparation (small sample of vaginal secretions to which drop of saline has been added) and are regarded as pathognomonic of bacterial vaginosis. The most objective way of identifying the clue cell is to observe the cell borders. If the vaginal cell border has a serrated appearance and cannot be identified clearly because of the attachment of large number of bacteria, a clue cell is present. The appearance of a dirty, hazy or cloudy interior of the epithelial cell is

more subjective in identifying a clue cell than are criteria utilizing the cell border.

It is not necessary to see clue cells to make the diagnosis of bacterial vaginosis, as the key feature is the reduced or absent Gram-positive large rods of Lactobacilli, and their replacement by Gram-variable or Gram-negative rods. Atleast there should be 20% of epithelial cells having the appearance or clue cells in a wet mount of vaginal fluid (Escherbach et al., 1998) to diagnose bacterial vaginosis.

Recognition of clue cells which is an excellent predictor of bacterial vaginosis is subject to variability. Because of these drawbacks a simple, inexpensive method for diagnosis of Bacterial vaginosis was assessed. Gram stain of the vaginal fluid has been used for confirmation of bacterial vaginosis since 1965.

GRAM STAIN

It has been demonstrated that Gram stain diagnosis alone corresponds well to the use of composite criteria and to the presence of the associated bacteria. It is a more objective method of diagnosis. The slides can also be stored for future reference. Patient-collected 'blind' vaginal swabs have been demonstrated to be as accurate as

swabs taken using a speculum, Gram stains are the only method of diagnosing an intermediate category of vaginal flora which is not as dramatic as bacterial vaginosis but is still abnormal.

Diagnosis is made on the basis of presence of Gardnerella morphological types out numbering lactobacilli and presence of other bacterial morphologic types. Gram stain has a greater utility in diagnosing the condition.

Gram stain interpretation of Spiegel et al., 1983

Less than one organism per field	1+
1-5 organisms per field	2+
6-30 organisms per field	3+
>30 organisms per field	4+

Presence of large number of Gram positive lactobacilli morphology alone or greatly exceeding other morphological types is labeled as negative Gram stain for bacterial vaginosis. When lactobacilli morphological types are present at >2+ levels and there are 3+ to 4 + levels of mixed flora including Gardnerella cocci, rods, fusiform bacteria or curved rods slides are interpreted as positive for bacterial vaginosis.

NUGENT SCORE

Patients were considered to have bacterial vaginosis by the Nugent Gram stain method if the following criteria were met. Numerical values were assigned to each quantification of morphotypes and a scoring system from 0-10 for grading the severity of bacterial vaginosis.

Nugent Score :

S.No.	Bacterial morphotype	None	1+	2+	3+	4+
1.	Large Gram positive rod (Lactobacilli)	4	3	2	1	0
2.	Small Gram negative / variable rod (Gardnerella, Bacteriods)	0	1	2	3	4
3.	Curved Gram negative / variable rod (Mobiluncus)	0	1	1	2	2

0 – 3 Negative

4 – 6 Intermediate

$\geq$ 7 Bacterial vaginosis

Thomason et al attempted to combine the criteria of Amsel et al and noted that lactobacilli morphologic types could be determined in wet mount examination without the use of gram staining

Criteria of Thomason et al

1. Thin homogenous vaginal discharge
2. Vaginal pH>4.5
3. Presence of clue cells
4. Release of fishy odor (amine test) after addition of 10% KOH to the vagina discharge.
5. Non lactobacilli morphological types greater than lactobacilli morphological types in wet mount examination.

They claimed that bacterial vaginosis was present if four of the five criteria were met.

The main difficulty for obstetricians and gynecologists is the lack of instant access to direct microscopy which is the most reliable method of diagnosing bacterial vaginosis. A roll of narrow range pH paper is cheap and a normal vaginal pH virtually excludes bacterial vaginosis. The whiff test is also cheap and easy to do with high sensitivity and good specificity. New rapid tests for bacterial

vaginosis have been developed which measure metabolic products from anaerobic bacteria such proline aminopeptidase or are based on DNA probes, for example Affirm VPIII which probes for G.vaginalis genes. If a simple but accurate test similar to urine pregnancy tests could be developed for the diagnosis of bacterial vaginosis in the 'office gynecology' or antenatal clinic setting this could be an enormous advance in the clinical setting.

Special diagnostic method for bacterial vaginosis

1. Papanicolaou smear

clue cells and changes in bacterial flora can be found in the Papanicolaou smear, which normally would be incidental finding and has a limited diagnostic potential in comparison with other methods. Studies have shown to have a sensitivity of 90% and a specificity of 97%. Sehnading et al reported excellent correlation between Pap smear and Gram smear for diagnosis of bacterial vaginosis.

Culture

Cultures generally play no role in the diagnosis because the isolation of G.vaginalis and/or anaerobic bacteria from the vagina

does not define the clinical entity and can be observed in women without bacterial vaginosis.

Because bacterial vaginosis results from an overgrowth of multiple vaginal organisms, it is not suprising that culture is the least accurate means of defining the syndrome. Thus the isolation of G. vaginalis should not be used to establish a diagnosis or a cure. Culture techniques used to identify other anaerobic bacteria including the newly described mobiluncus species are expensive and to date, have not been necessary for diagnosis.

Proline amino peptidase activity

Thomason JL et al described aminopeptidase activity as a rapid diagnostic test to confirm bacterial vaginosis. This test is based on the detection of enzymatic activity. In this assay, the enzyme in the vaginal fluid cleaves the substrate L-proline β naphthylamide and release naphthylamine. The test requires no sophisticated instrumentation that can cause varying results from one laboratory to another, is highly specific and has a greater that 80% sensitivity. This test was found to be superior to gas-liquid chromatography for confirming the clinical diagnosis of bacterial vaginosis.

Gas-liquid chromatography

Gas liquid chromatogtraphy, can be used in the diagnosis of bacterial vaginosis. Organisms produce organic acids as byproducts of their metablism and this test can identify these metabolic organic acids. Each genus has a typical pattern of organic acid production and this pattern can be used to identify specific organisms. The presence of various fatty acids other than acetic or lactic acid (for example, propionic, butyric, isobutyric, succinic) has correlated well with the presence of bacterial vaginosis.

DIFFERENTIAL DIAGNOSIS OF VAGINAL INFECTIONS

Diagnostic criteria	Normal	Bacterial vaginosis	Candida vulvovaginitis	Trichomonas vaginitis
Vaginal pH	3.8 to 4.2	>4.5	>4.5	>4.5
Discharge	White flocculant	Thin, homogenous white, grey, adherent, often increased	White, curdy, cottage cheese like, sometime increased	Yellow, green, frothy, adherent, increased
Amine odour	Absent	Present (fishy)	Absent	Often present (fishy)
Micro scopic	Lacto bacilli	Clue cells, coccobacillary bacteria No white cells	Mycelia, budding yeast, pseudohyphae with KOH preparation	Trichomonas, WBC >10 per high power field.
Common patient complaints	None	None Discharge fishy odor possibly worse after intercourse	Itching/burning discharge	Frothy discharge odor, vulvar pruritus dysuria

COMPLICATIONS

Obstetric complications associated with bacterial vaginosis

spontaneous preterm labour (SPTL) and preterm birth (PTB)

The aetiology of PTB is multifactorial, but there is now well-accepted evidence to implicate infection as a cause in up to 40% of cases.The mechanism by which bacterial vaginosis can induce PTB is linked to ascending genital tract infection, with an immune response resulting in the production of proinflammatory cytokines such as interleukin-1 α (IL-1 α), interleukin - 1β (IL-1β) and tumour necrosis factor- α (TNF- α). Cytokines are proteins secreted during inflammatory processes with an immunological basis and play a role in intercellular signaling. They are present during the process of normal labour, but higher concentrations have been found in the aminotic fluid of women in SPTL due to infection, which sets off a cascade resulting in the recruitment of inflammatory mediators such as prostaglandins. This eventually leads to cervical ripening and uterine contractions that may result in preterm labour. Phospholipase A_2 (PLA_2) and phospholipase C (PLC) are enzymes responsible for cleaving arachidonic acid, the obligate precursor for prostaglandin synthesis, from glycerophospholipids in the cell

membrane and have been found to be elevated in the lower genital tract of women with bacterial vaginosis.

Inflammation of the choriodecidual space cause release of fibronectin. Detection of fibronectin in cervicovaginal secretions after 22 weeks gestation is predictive of preterm delivery and associated with bacterial vaginosis. Phosphorylated insulin-like growth factor binding protein is produced by inflamed deciduas and can be detected as early as 8 weeks gestation and may therefore a better marker for adverse pregnancy outcome

Approximately 15-20% of all pregnant women will have bacterial vaginosis and these women are up to 4 times more likely to have a PTB than women without bacterial vaginosis. In a longitudinal study, Hillier et al., demonstrated that women with bacterial vaginosis are 40% more likely to deliver a

preterm, low birth weight infant than women without bacterial vaginosis.

Women with bacterial vaginosis in the second trimester tended to remain bacterial vaginosis positive in the third trimester and those women with intermediate flora had a significant chance of progressing to bacterial vaginosis. In a longitudinal study by Hay et

al., the Gram-stained, vaginal smears of 718 pregnant women were examined for bacterial vaginosis until 36 weeks' gestation. The results showed that, of those women who initially had normal flora at their first antenatal visit, only 2.4% developed bacterial vaginosis by 36 weeks' gestation. Of 32 women who had bacterial vaginosis initially, half had abnormal vaginal flora by 36 weeks.

Late miscarriage

The incidence of "late miscarriage (13 -23 weeks' gestation) has been demonstrated to be significantly higher in women who have bacterial vaginosis than those who do not, and to be independent of other risk factors. Since late miscarriage is on a continuum with extremely early preterm birth, the mechanisms by which bacterial vaginosis is associated with late miscarriage are assumed to be similar to those for PTB.

Postpartum endometritis

Postpartum endometritis following a Caesarean section tends to develop within 2 days and is described as early endometritis. This is most likely to be due to the introduction of bacteria into the endometrial cavity at delivery. Women who have a vaginal delivery usually develop late endometritis, which can occur up to 6 weeks

post- natally. This delayed infection tends to result from ascending infection over a course of time. Facultative anaerobes linked to bacterial vaginosis commonly isolated in late endometritis. Women with bacterial vaginosis were nearly 6 times were likely to develop the condition than women without BV.

GYNAECOLOGICAL COMPLICATIONS ASSOCIATED WITH BACTERIAL VAGINOSIS

Bacterial vaginosis and cervical intra-epithelial neoplasia (CIN)

A possible association between bacterial vaginosis and CIN has been explored by various studies over many years. The relationship between bacterial vaginosis and cervical dysplasia/carcinoma is inconsistent, as other studies have found no relationship between bacterial vaginosis and CIN /cervical carcinoma. The main criticism of previous studies investigating the possible role of bacterial vaginosis in the aetiology of CIN is failure to control for sexually transmitted infections, particularly oncogenic human papilloma virus (HPV) infection – a known risk factor for cervical neoplasia.

It has been suggested that some vaginal flora, such as the anaerobes associated with bacterial vaginosis, are capable of

producing carcinogenic substances called nitrosamine. Evidence regarding production of nitrosamine by bacterial vaginosis organisms is conflicting and the proposed mechanism of action by which nitrosamine may act has also been ill-defined although through to be by exerting an influence via enhanced replication of oncogenic HPV.

Pelvic inflammatory disease (PID)

In the past, PID was commonly caused by Chlamydia trachomatis or N.gonorrhoeae , but current attention is focused on the effects of bacterial vaginosis related micro-organisms. Cervicovaginal fluids can be sucked through the cervix into the uterus and beyond during spontaneous / hormone-mediated uterine contractions at mid-cycle. Due to polymicrobial nature of bacterial vaginosis it is difficult to attribute bacterial vaginosis associated PID to any one organism.

Infertility and first trimester loss

Several studies have examined the possible relationships between bacterial vaginosis and infertility. Although in one study, a higher prevalence of bacterial vaginosis was found in women undergoing invitro fertilization (IVF) than in the general population,

other studies have not found this to be the case, except in women whose infertility was attributable to tubal disease. A recent UK study found that there was a high prevalence of bacterial vaginosis in women undergoing IVF and that women with bacterial vaginosis had a higher rate of first trimester miscarriage than those with normal vaginal flora. Most of these losses were in 'chemical' pregnancies in the bacterial vaginosis group. There is speculation that bacterial vaginosis related bacteria may have an adverse effect on sperm deposited in the vagina and thus reduce fertility, although this has been disputed.

Post-hysterectomy vaginal cuff infection

post- hysterectomy vaginal cuff infection occur 3-4 times more commonly in women with bacterial vaginosis than in those without. The use of prophylactic antibiotics to prevent vaginal cuff infection is now generally an accepted practice, but wide-spread use of different antibiotics may not eradicate bacterial vaginosis and its related organisms but lead to greater resistance.

Postabortal sepsis

First trimester surgical termination of pregnancy remains a common procedure in gynecological practice. Postoperative

infection, such as endometritis, occurs at rates between 4-12% Pelvic infection following termination of pregnancy may be due to vaginal infections particularly with N.gonorrhoeae, C.trachomatis and bacterial vaginosis –related organisms. The use of antibiotic prophylaxis before surgical termination of pregnancy demonstrates a protective effect. There is strong evidence to suggest that women should preferably be screened and treated for bacterial vaginosis as well as other infections, such as C.trachomatis, prior to termination of pregnancy or given appropriate prophylactic antibiotics.

Urethral syndrome

Urethral syndrome can be defined as dysuria in women that cannot be explained by the bacteria that normally cause urinary tract infection. C.trachomatis has been implicated in some cases. BV is also implicated in the aetiology of urethral syndrome. This theory needs further investigation.

Bacterial vaginosis and acquisition of human immunodeficiency virus (HIV)

Klebanoff et al., showed tht the presence of hydrogen-peroxide-producing Lactobacilli in the vagina results in a more acidic environment which is not only toxic to bacterial vaginosis

associated flora but also to HIV. They postulated that a lower vaginal pH may block the production of CD4 lymphocytes whereas a higher, more alkaline pH associated with bacterial vaginosis, may enhance HIV survival.

M.hominis increase the activity of a soluable HIV inducing factor (HIF) and therefore increase HIV-1 expression. Genital tract infection with G. Vaginalis stimulate HIV-1 production and hence increase the likelihood of sexual transmission.

THERAPY

The polymicrobial nature of bacterial vaginosis poses a problem to clinicians in attempting to find the most appropriate drug therapy. Currently, treatment recommendations world wide advocate that bacterial vaginosis may be treated with either metronidazole or clindamycin, given either orally or vaginally.

Treatment of bacterial vaginosis

			Cure rate
1.	**Metronidazole**	500 mg bid x 7 day	70-100%
		2 gms stat dose	65-80%

Metronidazole is not advised during pregnancy especially in I trimester (? Carcinogenic effect).

2. **Clindamycin** 300 mg bid x 7 days _ Cure rate 94%

INDICATIONS

- in the treatment of bacterial vaginosis during pregnancy.
-In metronidazole treatment failure
-In patients who cannot tolerate metronidazole.

3. **Ampicillin** active against G. Vaginalis however resistant beta lactamase producing strains of one or more Prevotella may be present in bacterial vaginosis.

- Ampicillin also kills lactobacilli
- So cure rate is 43% in the treatment of bacterial vaginosis.

4. **Amoxycillin** with clavulanic acid (Augmentin) may be useful, but inhibits recolonization of lactobacilli.

5. Other antibodies – ciprofloxacin, cephalexin, tetracycline and Erythromycin are less effective.

Intra vaginal therapy :

1. Intravaginal metronidazole 500 mg for 7 days was compared with 400 mg orally bid x 7 days. Cure rate was 79% for the intra vaginal therapy compared with 74% for oral therapy. Usual metallic taste, headache, gastro intestinal distresss

associated with oral metronidazole is absent with intra vaginal gel application.

2. Clindamycin cream 5 gm in 0.1%, 1.0%, 2.0% strengths twice a day for 7 days – is also effective. With 2% cream – the recurrence rate was low. Once a day application 0.5g of clindamycin 2% cream for 4-7 days has a cure rare of 94%.
3. Vaginal douching with povidone iodine, application of acetic acid gel, dienestrol cream, triple sulfa cream are not effective.
4. Recently lactate gel containing lactic acid and growth substrate for lactobacilli buffered to pH 3.8 has been tried and produced a high cure rate. One unit of 5 ml of the gel is applied intravaginally every night for 7 days. After 2 days of treatment lactobacilli reappear and are the predominant organism.

Self – help :

The avoidance of washing the genital area with soap, shower gel or other alkaline detergents will help prevent bacterial vaginosis. pH balanced soaps for washing are still considerably more alkaline than the vagina and may, therefore promote the emergence of bacterial overgrowth.

The practice of douching should be discouraged. The use of natural yoghurt or Lactobacillus acidophilus provide short term relief. Products containing Lactobacillius spp. are widely available and are used in an attempt to restore normal flora. Hillier demonstrated the effectiveness of a vaginal capsule containing Lactobacillus crispatus for colonization in 90 sexually active adolescent young women. Capsules contained either 10^6 or 10^8 L. crispatus and were inserted twice daily for 3 days ; assessment carried out weekly for one month. In the women who had significantly reduced numbers of hydrogen peroxide – producing Lactobacilli prior to use of the capsule, 76-85% of follow up visits showed sustained colonisation at assessment. It appears to be some potential value in the use of exogenous strains of some Lactobacilli.

Treatment of male partners :

Most trails involving treatment of the male partner for bacterial vaginosis have not resulted in any improvement in the cure rate in the female. A recent appraisal of six trials assessing the treatment of the male sexual partner of women with bacterial vaginosis suggests that there appears to be no benefit in doing so. The general consensus currently appears to be that there is no

justification in treating the male partner of a women with bacterial vaginosis.

TREATMENT FAILURE

In treatment failure cases, the vaginal swab to be taken from the lateral walls and should be sent for culture in an anaerobic transport medium. A broad spectrum agent to be chosen whose main strength is directed against the dominant organism. The patients should be reevaluated within two weeks of completing therapy.

MATERIALS AND METHODS

The present study was carried out on women admitted in the labor ward in the Department of Obstetrics and Gynaecology, Government Rajaji Hospital, Madurai Medical College, Madurai.

A total number of 200 women were studied which were divided into 2 groups.

The study group included 100 women who came in preterm labor ie.

- With gestational age between 28 and 37 weeks
- With painful uterine contractions lasting for 45 seconds associated with cervical effacement of 80% and above
- Cervical dilatation of less than or equal to 3 cm and with intact membranes.

The control group included 100 women who came in term gestation in labor.

- With painful uterine contractions for 45 seconds associated with cervical effacement of 80% and above.
- Cervical dilatation of less than or equal to 3 cm and with intact membranes.

Inclusion Criteria

- Booked, unbooked and referral cases were included in the study
- Both primi and multi irrespective of socio economic status were included.

Exclusion criteria :

Women are excluded from analysis if they had

- GA < 28 weeks
- Multiple pregnancy
- Malpresentation
- Placenta previa / APH
- Cervical incompetence treated with cervical encerlage
- Hydramnios
- Pregnancy induced hypertension
- Fever, UTI, Diarrhea, Respiratory tract infection
- Anaemia, Heart disease, GDM, DM
- PROM / absent membranes
- Antibiotic therapy within last 30 days
- Intra uterine growth retardation
- Intra uterine death

Clinical study :

A complete history was taken with menstrual history and obstetrical history. The gestational age was confirmed from last menstrual period and was correlated with clinical examinations and ultrasonographic gestational age. In the case of previous history of preterm labour the ultimate fetal and maternal prognosis was carefully analysed. In the current pregnancy a detailed history of complication associated with pregnancy was taken.

Abdominal vaginal and speculum examination were done. Nature of discharge noted and vaginal swabs were taken for bacteriologic study.

Bacteriological study :

The specimen was collected by putting the patient in dorsal supine position. Under all aseptic conditions the posterior vaginal wall was retracted with sims speculum and vaginal swabs were taken from posterior fornix by 3 sterile cotton swabs.

pH test :

By using a piece of nitrazine paper, pH of the vaginal fluid can be obtained. Care was taken to avoid contact with cervical mucus, as the pH of cervical secretions is approximately-7.

Amine Test :

A drop of 10% KOH was added to wet mount specimen and fishy odor was noted.

Clue cells on wet mount :

Clue cells are found by mixing vaginal fluid with a drop of normal saline on a slide and examining this slide microscopically under high power magnification (x400). Specimens were considered adequate if at least 10 epithelial cells per high power field were seen. The presence of even as few as one clue cell per field in 20 fields (x400) was considered positive. Clue cells were identified as vaginal epithelial cell with indistinct cell border obscured by the large number of attached organisms.

AMSEL criteria

Amsel et al claimed that bacterial vaginosis was present if three of these four criteria was present.

1. Homogenous vaginal discharge
2. Vaginal pH > 4.5
3. Fishy odour on alkalinization of vaginal secretion.
4. Presence of clue cells

Gram Staining :

With the swab obtained from the posterior fornix a direct smear was made on a clean slide and gram staining was done and smear examined for the presence of clue cells gram negative coccobacilli and other morphological types.

Method of gram staining :

To a dried smear

Step I : Methyl violet was added, washed with tap water after one minute

Step II : Grams iodine was added, washed with tap water after one minute

Step III : Acetone was added, washed with tap water after 30 seconds

Step IV: Dilute carbol fuschin was added, washed with tap water after one minute

Step V : Smear air dried, study of gram stained smear was done in x 1000 magnification with oil immersion.

The presence of clue cells and the number of lactobacilli and other morphological types were noted. The results were interpreted

using spiegels. In this method the number of organisms per high power field are graded as

< 1 per field 1 +

1 – 5 per field 2+

5 – 30 per field 3 +

> 30 per field 4+

Large gram positive bacilli are assumed to be lactobacillus morphotypes and smaller gram variable coccobacilli to be gardnerella morphotypes.

Presence of large number of gram positive lactobacilli morphology alone or greatly exceeding other morphological types is labeled as negative gram stain for bacterial vaginosis.

When lactobacilli morphological types are present at < 2+ levels and if there are 3 + to 4+ levels of mixed flora including Gardnerella cocci, rods, fusiform bacteria or curved rod slides are interpreted as positive for bacterial vaginosis. (Spiegel et al)

Nugent score :

S.No.	Bacterial morphotype	None	1+	2+	3+	4+
1.	Large Gram positive rods(Lactobacilli)	4	3	2	1	0
2.	Small Gram negative / variable rod (Gardnerella, Bacteriods)	0	1	2	3	4
3.	Curved Gram negative / variable rod (Mobiluncus)	0	1	1	2	2

0 – 3 Negative

4 – 6 Intermediate

$\geq$7 Bacterial vaginosis

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2008).**

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

Sensitivity, specificity, accuracy, positive predictive value and negative predictive values were calculated using the following formulae and taking 48 hours positivity results with 10 mm induration as cut off value as the Golden standard.

$$\text{Sensitivity} = \frac{\text{True positive}}{\text{True positive + False negative}} \times 100$$

$$\text{Specificity} = \frac{\text{True negative}}{\text{False positive + True negative}} \times 100$$

$$\text{Accuracy} = \frac{\text{True Positive + True Negative}}{\text{Total cases}}$$

$$\text{Positive predictive value} = \frac{\text{True positive}}{\text{True positive + False positive}} \times 100$$

$$\text{Negative predictive value} = \frac{\text{True negative}}{\text{True negative + False negative}} \times 100$$

OBSERVATIONS, RESULTS AND ANALYSIS

Having excluded patients with known risk factors for preterm labour, the subset of women in idiopathic preterm labour (100) and women in term labours (100) were studied. Nugent's score (Gram staining) taken as gold standard test to diagnose bacterial vaginosis

Table -1 : Age Distribution of Cases

Age groups (in years)	Pre term	Term
< 20	9	9
21 – 24	56	50
25 – 28	22	33
29 – 32	9	6
33 Yrs & above	4	2
Total	100	100
Range	18 – 36 yrs	17 – 36 yrs
Mean	23.6 yrs	23.8 yrs
S.D	3.8 yrs	3.5 yrs

Mean Age in preterm group is 23.6 years $\pm$ 3.8 years and in term group is 23.8 years $\pm$ 3.5 years.

X^2 - 0.5843.

P - 0.4445 (not significant)

On analyzing the table, maternal age did not seem to influence the study group of idiopathic preterm labour as the percentage of case in the study group and the control group did not vary much.

Table -2

Distribution of subjects according to socio economic status

Socio economic status	Pre term	Term
Class I	0	0
Class II	0	0
Class III	5	17
Class IV	40	55
Class V	55	28

None of the patients studied were in class I & II Socio economic class. 55% of the study group belonged to class V compared to 28% of the control group.

X^2 - 13.92

P - 0.0002 which is statistically significant.

Table -3

Distribution of subjects according to obstetric history

Obstetric Code	Study (Pre term)	Control (Term)
Primi	57	45
G2	24	39
G3	14	13
G4	5	2
G5 & above	0	1

57 of women in preterm group and 45 in term group were primigravidas. 5 cases in preterm group and 2 cases in term group belonged to G4. 1 case in term group belonged to G6 and none in the preterm group belonged to G5 & above.

X^2 - 2.42

P - 0.1197

The comparison was not significant

Table -4

Distribution of Cases according to Antenatal Care

Antenatal Care	Study (Pre term)	Control (Term)
Booked	40	49
Unbooked	60	51

- 40 cases in preterm group and 49 cases in term group were booked cases
- 60 cases in preterm group and 51 cases in term group were Unbooked

X^2 - 1.3

P - 0.255

Hence, comparison was not significant.

Table -5

Distribution of subjects according to their weight

Weight (in kg)	Study (Pre term)	Control (Term)
41 – 45	3	1
46 – 50	33	3
51 – 55	40	9
56 – 60	15	28
61 – 65	4	27
66 – 70	1	16
> 70	4	16
Range	42 -84	44 – 82
Mean weight	53.7	63.1
S.D	7.2	7.5

No.of women < 50 kgs

In study group 36

In control group 4

X^2 - 78.02

P - 0.0001

which is statistically significant

women < 50 kg were 9 times more prone for preterm labour.

Table -6

Distribution of Bacterial vaginosis among the subjects

Bacterial vaginosis	Study (Pre term)	Control (Term)
Positive	27	12
Negative	73	88

According to Nugent's score Bacterial vaginosis was positive in 27% of preterm and 12% of term cases.

X^2 - 6.24

P - 0.0125

which is statistically significant. So the presence of Bacterial vaginosis was significantly associated with preterm labour.

Table -7

Distribution of subjects according to past obstetric history

Previous obstetric history	Study (Pre term)	Control (Term)
Previous abortion		
a. spontaneous	12	10
b. Induced	1	1
Previous preterm delivery	7	1

13% of preterm patients and 11% of term cases had previous history of aortions.

X^2 - 0.05

P - 0.8233

which is not significant statistically

7% of preterm cases and 1% of term cases had previous preterm deliveries.

X^2 - 3.26

P - 0.0326

Hence the comparison is significant. Patients with previous preterm deliveries were more prone for subsequent preterm deliveries.

Table -8

Efficacy of various investigations / Homogenous Discharge

Homogenous discharge	Bacterial Vaginosis	
	Positive (n=39)	Negative (n = 161)
Present (n=36)	31	5
Absent (n=164)	8	156

	Percentage
True positive	86
False positive	14
True negative	95
False negative	5
Sensitivity	79
Specificity	94
Accuracy	94
PPV	86
NPV	95

36 cases (including term and preterm) had homogenous discharge characteristic of bacterial vaginosis. Of these, 31 had bacterial vaginosis according to Nugent's score.

The sensitivity was 79% specificity was 94% positive predictive value 86% negative predictive value 95%.

Table -9

pH values of Vaginal Discharge

Ph > 4.5	Bacterial Vaginosis	
	Positive (n=39)	Negative (n = 161)
Present (n=67)	39	28
Absent (n=133)	0	133

	Percentage
True positive	58.2
False positive	41.8
True negative	100
False negative	0
Sensitivity	100
Specificity	83
Accuracy	86
PPV	58
NPV	100

67 cases had pH > 4.5. Of these only 39 cases were positive for bacterial vaginosis. pH value was sensitive to diagnosis all cases of BACTERIAL VAGINOSIS. The sensitivity was 100%. Specificity was 83%. positive predictive value was only 58%. Negative predictive value was 100%.

Table -10

Amine Test Findings

Amine Test	Bacterial Vaginosis	
	Positive (n=39)	Negative (n = 161)
Present (n=44)	37	7
Absent (n=156)	2	154

	Percentage
True positive	84.1
False positive	15.9
True negative	98.7
False negative	1.3
Sensitivity	95
Specificity	96
Accuracy	96
PPV	84
NPV	99

44 cases were positive for amine test of these 37 cases were positive for bacterial vaginosis according to Nugent's score.

The sensitivity was 95%. specificity 96%. Positive predictive value 84%, Negative predictive value 99%.

Table -11

Findings of clue cells

Clue cells	Bacterial Vaginosis	
	Positive (n=39)	Negative (n = 161)
Present (n=32)	30	2
Absent (n=168)	9	159

	Percentage
True positive	93.8
False positive	6.3
True negative	94.6
False negative	5.4
Sensitivity	77
Specificity	99
Accuracy	95
PPV	94
NPV	95

32 cases were positive for clue cells. Of these 30 cases had BV according to Nugent's score. 9 cases who were negative for clue cells had bacterial vaginosis according to Nugent's score.

The sensitivity was only 77%, specificity was 99% positive predictive value was 94% Negative predictive value was 95%.

Table -12

Findings of Amsel's criteria

Amsel's criteria	Bacterial Vaginosis	
	Positive (n=39)	Negative (n = 161)
Present (n=37)	37	0
Absent (n=163)	2	161

	Percentage
True positive	100
False positive	0
True negative	98.8
False negative	1.2
Sensitivity	95
Specificity	100
Accuracy	99
PPV	100
NPV	99

100% cases who were positive for BV by Amsel's criteria had bacterial vaginosis by Nugent's score. Amsel's criteria did not have any false positive cases. But this criteria failed to diagnose bacterial vaginosis in 2 cases who were positive for bacterial vaginosis according to Nugents score. The sensitivity was 95%. specificity 100%. positive predictive value 100%, Negative predictive 99%.

Table -13

Efficacy of various test results

Tests	Sensitivity	Specificity	Accuracy	PPV	NPV
Nature of discharge	79	94	94	86	95
pH > 4.5	100	83	86	58	100
Amine test	95	96	96	84	99
Clue cells	77	99	95	94	95
Amsel's criteria	95	100	99	100	99

On analyzing various test results pH value > 4.5 has highest sensitivity (sensitivity 100%)

Amine test, Amsel's criteria and clue cells have high specificity

Amsel's criteria - 100% specific

Clue cells - 99%

Amine test - 96%

Amine test & Amsel's criteria have good sensitivity and specificity and correlate well with Nugent's score.

Table -14

Impact of Bacterial vaginosis on mode of delivery

Mode of delivery	Bacterial Vaginosis	
	Positive (n=39)	Negative (n = 161)
LN (n=180)	34	146
Outlet (n=18)	5	13
LSCS (n=2)	0	2

Of those 39 patients who were positive for bacterial vaginosis 34 patients delivered by labour natural and 5 patients delivered by outlet forceps.

Out of 161 patients who did not have bacterial vaginosis 146 patients delivered by labour natural, 13 patients delivered by outlet forceps and 2 patients were delivered by LSCS.

From the table the presence of bacterial vaginosis did not seem to increase the rate of instrumental deliveries and caesarean deliveries.

X^2 - 0.13

P -0.345

Hence the comparison was not significant.

Table -15

Distribution of bacterial vaginosis according to the birth weight of the baby

Birth weight (in Kg)	Bacterial vaginosis	
	Positive (n=39)	Negative (n = 161)
1 – 1.5	6	7
1.6 – 2	10	20
2.1 – 2.5	12	56
2.6 – 3.0	9	44
> 3 kg	2	34
Range	1.25 – 3.5	1 – 3.8
Mean	2.21	2.53
S.D	0.55	0.57

The mean birth weight of babies in the bacterial vaginosis. Positive group was 2.21 kg + 0.55 kg. where as in bacterial vaginosis Negative group was 2.53+ 0.57

X^2 = 9.1949

P = 0.0024

On analyzing the data the presence of bacterial vaginosis was significantly associated with low birth weight babies.

Table – 16

Impact of bacterial vaginosis on maternal and neonatal outcome

Maternal and neonatal complications	Bacterial vaginosis				X2	P value
	Positive (n=39)		Negative (n=161)			
	No.	%	No.	%		
Neonatal complications						
Present	9	23.1	22	13.6	1.47	0.226 (Not significant)
Absent	30	76.9	139	86.4		
NICU Admissions						
Present	16	41	27	16.8	9.55	0.0019
Absent	23	59	134	83.2		Significant
Maternal Complications						
Present	2	66.77	1	33.3	1.8	0.978 (not significant)
Absent	37	19	160	85		

From the table, the neonatal complications (including birth asphyxia, RDS, meconium aspiration syndrome, hyperbilirubinemia) were not increased in patients with bacterial vaginosis when compared with patients who did not have bacterial vaginosis.

X^2 - 1.47

P - 0.226 (not significant)

NICU admissions in bacterial vaginosis positive group was 41 % whereas in bacterial vaginosis negative group the NICU admissions were only 16.8%.

X^2 - 9.55

P - 0.0019

which was statistically significant.

2 patients in bacterial vaginosis positive group had episiotomy wound infection where as in bacterial vaginosis negative group 1 patient had puerperal fever.

X^2 - 1.8

P - 0.978 which was statistically not significant.

DISCUSSION

From the study we have confirmed significant association between bacterial vaginosis and preterm labour.

Gram staining and analysis by Nugent scoring has been taken as a standard method of diagnosing Bacterial vaginosis in our study because of the reliability and accuracy of Nugent score in the detection of bacterial vaginosis Nugent's score also has the advantage of less inter and intraobserver variation, and the slides can be stored for future references.

In our study the prevalence of bacterial vaginosis was 27% in the study group of preterm labour and in 12% in the control group of term labour (p - 0.012)

Our study corresponds to Holst et al (2007), Sharon et al (2004) and Thanavuth et al (2007).

Study	Study group	Control Group
Holst et al (2007) Dept. of microbiology lund university Sweden	31%	11%
Chaijarcenont et at (2004) J. Med assoc. Thai	36%	8%
Hilmars dottir et al (2006) J. Clin, Microbiology	15%	5%

Sharon et al (2004) University of Pittsburgh, PA	32%	14%
Thanavuth et al (2007) Deportment of O.G. Siriraj hospital, Mahidol University	28%	9%
Our study	27%	12%

In our study, mean age in study group was 23.6 yrs $\pm$ 3.8 yrs and in control group was 23.8yrs $\pm$ 3.5 yrs. The age distribution of cases in the study and control group did not vary much which corresponds to that of Iams et al (1999) and to that of MC Donald et al (1991)

In our study, there was a significant association of women in very low socio economic status (Class V) and preterm labour (p – 0.0002). This corresponds to that of a study conducted at the department of microbiology, Government Medical college, patiala Punjab (2001).

In our study, the distribution of cases according to the parity in the study and control group did not vary much (p – 0.1197) which is in accordance to Robert L Goldenberg et al (2002).

In our study, the mean maternal weight in study group was 53.7 + 7.2 kg whereas in control group the mean maternal weight

was 63.1 + 7.5 kg. Out of 100 patients in the preterm group 36 (36%) patients were < 50 mg whereas in term group only 4 patients (4%) were < 50 kg. So there was a significant association between maternal weight less than 50Kg and preterm labour (p – 0.0001). This corresponds to the study of Andrews WW et al (2001) Birmingham Alabama.

In our study, there was significant association of previous preterm birth (7%) to preterm labour (p – 0.0324) this corresponds to that of Cunningham et al (2005).

S.No.	Study	Study group	Control group
1	MC Donald et al (1994)	17%	4%
2	MC Gregor et al (2001)	30%	6.8%
3	Cunningham et al (2005)	10%	2%
4	Present study	7%	1%

On analyzing efficacy of various tests homogenous discharge was present in 36 patients of these 31 were positive for bacterial vaginosis. 8 cases of bacterial vaginosis positive cases did not have homogenous discharge the sensitivity was 79% specificity was 94%.

PH >4.5 found in 67 patients and diagnosed all of bacterial vaginosis positive cases with the false positivity rate of 41.8% pH>4.5 has the highest sensitivity (100%) and least specificity

However it is economical, extremely simple and a useful tool to rule out bacterial vaginosis.

Amine test was positive in 44 cases of which 37 cases were positive for bacterial vaginosis. Amine test has a good sensitivity (95%) and a specificity (96%). In the absence of microscope, amine test can be used as a specific and relatively sensitive method of detecting BV.

Detection of clue cell is the single most specific test but not a sensitive one specificity 99% sensitivity 77%. It has a high PPV (94%) and a NPV (95%).

We did not evaluate culture for G.vaginalis since it has repeatedly been shown to be of little diagnostic value. Eschenbach's group found that more than 55% of normal patients had G. vaginalis positive. Culture play no role in diagnosis because isolation of G.vaginalis and or anaerobic bacteria from vagina does not define the clinical entity and can be observed in women without bacterial vaginosis.

In our study, the mean birth weight of babies born to bacterial vaginosis positive mothers was 2.21 Kg.$\pm$ 0.55kg and in bacterial vaginosis negative group was 2.53$\pm$ 0.53kg (p – 0.0024). Hence BV

is significantly associated with LBW babies. Whether it is the cause (or) association is not known.

In our study, out of 39 patients who had bacterial vaginosis neonatal complications (birth asphyxia, RDS, meconium, aspiration syndrome, hyper bilirubinemia) were present in 9 cases (23.1%). In bacterial vaginosis negative group out of 161 patients in 22 babies (13.6%). So the incidence of neonatal complications in bacterial vaginosis positive and negative group did not vary much (p - 0.226) in our study.

In our study, out of 39 patients who had bacterial vaginosis 16 babies (41%) were admitted in neonatal intensive care unit whereas in bacterial vaginosis negative group out of 161 babies, 27 babies (16.8%) were admitted in NICU. So the neonatal admissions in bacterial vaginosis positive group were higher (p – 0.0019) than the bacterial vaginosis negative group.

In our study, 4 babies in preterm group and 1 baby in term group died in the early neonatal period. None of these babies were in bacterial vaginosis positive group. So the presence of bacterial vaginosis did not seem to influence the neonatal outcome.

Among maternal complications only the infectious morbidity (episiotomy wound infection, fever, foul smelling discharge) was analysed. 1 patient in study group and 1 patient in control group had atonic PPH and 1 patient in control group developed lumbar plexopathy. All these complications were excluded from analysis.

2 Patients in bacterial vaginosis positive group developed episiotomy wound infection and 1 patient in bacterial vaginosis negative group developed fever in the puerperal period. The infectious morbidity in the bacterial vaginosis positive and negative group did not differ much (p – 0.0978).

Among 39 patients who had bacterial vaginosis 34 patients delivered by labor natural, 5 patients had outlet forceps deliveries and none of the patient had caesarean delivery. Where as in patients who did not have bacterial vaginosis (Total 161) 146 patients delivered by labor natural 13 patients delivered by outlet forceps and 2 patients delivered by LSCS.

The incidence of instrumental deliveries and caesarean deliveries did not vary much between bacterial vaginosis positive and negative group(p - 0.345).

SUMMARY

- 100 women in idiopathic preterm labour (study) and 100 women labour (control) were studied.
- Maternal age and parity did not seem to influence the study group
- There was significant association of women belong to very low socio economic status class V to the study group (p – 0.0002)
- There was significant association of maternal weight less than 50kg to the study group (p – 0.0001)
- There was significant association between previous spontaneous preterm delivery and study group (preterm group) (p – 0.0324)
- There was significant association between bacterial vaginosis and preterm labour (p – 0.0125)
- There was significant association between bacterial vaginosis and delivery of low birth weight babies (p – 0.0024).
- There was no increase in neonatal or maternal complications in bacterial vaginosis positive group

- There was no significant association between bacterial vaginosis and instrumental deliveries and caesarean deliveries (p – 0.345).
- Among various studies to diagnose bacterial vaginosis pH estimation has the highest sensitivity (100%) and presence of clue cells has the highest specificity (99%).
- Use of culture is of little diagnostic value in diagnosing bacterial vaginosis.

CONCLUSION

The prevalence of bacterial vaginosis was 27% in preterm group and 12% in term group. There was a significant association between bacterial vaginosis and preterm labour. There was also significant association of various factors like low socio economic status, low maternal weight and history of previous preterm deliveries to the study group (pre term labour). The neonatal and maternal outcome in the bacterial vaginosis positive and negative group did not differ much.

BIBLIOGRAPHY

1. Zarakolu P. Hodogiugil NNS, Aydin F, Tosun I., Gozalan A, Unal S. Diagno Microbiol Infect Dis 2004; 48:77-80.
2. E Holst; A.R Goffeng and B Andersch, Bacterial vaginosis and vaginal micro organisms in idiopathic premature labour and association with pregnancy outcome. Department of medical microbiology Lund Universities, Sweden J Clin Microbiol 2007, January; 32 (I): 176 – 186.
3. Chaijareenont et al 2004 J Med assoc. Thai
4. Nugent RP, Krohn MJ,Hillier S.L, Reliability of diagnosing bacterial vaginosis is improved by a standardized method of Gram stain interpretation. J Clin Microbiol 2001 i 29:1730 - 31.
5. Awassanan Thanavuth MD , Dittakarn Boriboohirutsam MD ,MPH, Phd, Ratre sirisomboon BSC , Kanjana pimst BN prevalence of bacterial vaginosis in Thai pregnant women with preterm lobour in Sriraj Hospital J Med Assoc Thai vol90 NoS2007.

6. Cunnigham FG, Lereno KJ, Bloom SL, Hauth JC, Glistrap LC 3rd , Wenstrom KD, preterm birth Williams obstetrics 22nd edition New York, MC – Grow – Hill 2005 ; 855 – 80
7. Sharon et al (2004) Association between bacterial vaginosis and preterm delivery of a Low birth weight infant, university of Pittsburgh, PA.
8. Gardner HL, Dukes CD, New etiologic agent in non-specific bacterial vaginosis, Sciene 1954;120:853
9. Gardner HL, Dues CD Hacmophilus vaginalis vaginitis: a newly defined specific infection previously classified 'non-specific' vaginitis Am j Obstet Gynecol 1955;69:962-976
10. Spiegel CA, Amsel R,Eschenbach DA et al. Anaerobic bacteria in non-specific vaginitis, N Engl J Med 1980;303:601-606
11. Eschenbach DA, Hillier SL, Critchlow CW et al. Diagnosis and clinical manifestations of bacterial vaginosis Am JObstet Gynecol 1988;158:819-828
12. Embree J, Caliando JJ, Mc Cormack WM. Non-specfic vaginitis among women attending a sexually transmitted discases clinic Sex Transm Dis 1984;11:81-84

13. Hay PE, Taylor-Robinson D,Lamont RF.Diagnosis of bacterial vaginosis in a gynaecology clinic. Br J Obstet Gynaecol 1992;99:63-66

14. Blackwell A, Thomas PD, Wareham Ket al. Health gains from screening for infection of the lower genital tract in women attending for termination of pregnancy Lancet 1993;342:206-210

15. Hay PE, Lamont RF, Taylor-Robinson D, Morgan DJ,Ison CA, PearsonJ Abnormal colonization of the genital tract and subsequent preterm delivery and late miscarriage BMJ 1994;308:295-298

16. Peters N, van Leeuvan AM, Peters W Bacterial vaginc sis is not important in the etiology of cervical neoplasia; a survey on women with dyskaryotic smears. Sex Transm Dis 1995;22:296-302

17. Amsel R, Totten P, Totten P, Spiegel CA Chen KC, Eschenbach DA, Holmes KK, Non-specfic vaginities: diagnostic criteria and microbial and epidemlological associations AmJ Med 1983;74:14-22

18. Klebanoff SJ, Hillier SL, Esche bach Da, Waltersdorf AaM.Control of the microbial flora of the vagina by H2o2 generating Lactobacilli J Infect Dis 1991; 154:94-100

19. Royce RA Jackson TP Thorp Jr J et al. Race/ethnicity, vaginal flora patterns and pH during pregnancy Sex Transm Dis 1999;26:96-102

20. Kinsman OS, Pitblado k, Coulson CJ, Effect of mammalian steroid hormones on the germination of candida albicans and implications for vaginal candidiasis. Mycosis 1988;31:617-626

21. Hay PE, Morgan DJ, Isen CA et al. A longitudinal study of bacterial vaginosis during pregnancy Br J Obstet Gynaecol 1994; 101;1048-1053

22. Vejtorp M, Bollerup AC, Vejtorp 1 et al. Bacterial vaginosis; a double-blind randomized trial of the effect of treatment of the sexual partner . Br J Obstet Gynaecol 1988;95.

23. Moi H,Erkkola R, Jerve F et al should male of women with bacterial vaginosis be treated? Genitourin Med 1989;65:263-268

24. Howe L, Wiggins R Soothil PW, Millar MR, Horner PJ, Coorfield AP.Mucinase and sialidae activity of the vaginal microflora: implications in the pathogenesis of preterm labour, Int J astd aids 1999;10442-447

25. Tao L, Pavlova S Mou Sm et al. Analysis of Lactobacilli products for phages and bacteriocins that inhibit vaginal Lactobacilli Infect Dis Obstet Gynecol 1997;5:244-251

26. Hillier SL Krohn M, Meyn Let al. Recolonisation of the vagina with an exogenous strain of Lactobacillus crispatus, Conference Proceeding of International Bacterial Vaginitis conference, Aspen, Colordo, USA, September 1998

27. Mc Cue JD.Evaluation and management of vaginitis. Arch Intern Med 1989;149:565-568

28. Hillier SL Krohn MA, Nugent RP, Gibbs RS Characteristics of three vaginal flora patterns assessed by Gram stain among pregnant women. Am Jobslet Gynecol 1902;166:938-944

29. Schwebke JR, Hillier sL, Sobel jD, Mc gregor JA, Sweet RL Validity of the vaginal Gram stain for the diagnosis of bacterial vaginosis. Obstect Gynecol 1996;88:573-576

30. Thomason JL, Gelbart SM, Wilcoski I.M. Peterson AK, Jilli Bj, Hamilton PR, Proline aminopeptidase activity as a rapid diagnostic test to confirm bacterial vaginosis Obset Gynecol 1988;71:607-611

31. Mc Gregor JA French JI Lawellin D et al. Preteyn birth and infection; pathogenic possibilities, Am J Reprod Immunol Microblol 2001;16:123-132

32. Mc Donald HM, O Loughlin JA Jolley PT et al Changes in "vaginal flora during pregnancy and association with preterm birth, J infect Dis 1994;170:724-728

33. Hillier SL, Witkin S, Krohjn M, Watts D, Kiviat N, Eschenbach DA. The relationship of amniotic fluid cytokines and preterm delivery, amniotic fluid infection, histologic chorioammionitis and chorioamnion infection. Obster Gynecol 1993;81:941-948

34. Mc Nichol P, Paraskevas M, Guijon F. Variability of polymerase chain reaction based detection of human papilloma virus DNA is associated with the composition of vaginal microbial flora. J Med Virol 1993;43:194-200

35. Goldenberg RL Mercer BM, Meis PJ, Copper RL Das A McNellis D.The preterm prediction study; fetal fibronectin testing and spontaneous preterm birth. Obstet Gynecol 1996;87:643-648

36. Kekki M, Kurki T, Paavonen J, Rutanen EM. Insulin-like growth factor binding protein I in the cervix as a marker of infectious complications in pregnant women with bacterial vaginosis (Letter) Lancet 1999;353;1494Frega A Stentella P, Spera g et al. cervical intraepithelial neoplasia and bacterial vaginosis; correlation or risk factor? Eur J Gynaecol Oncol 1997;18:76-77

37. Parsons A, Mc Gregor JA. Pathogens on the move: active uterine transport of cervicovaginal fluid into the uterus and fallopian tubes at ovarian mid-cycle . Int J astd aids 1997;8:22-22

38. Taylor-Robinson D Non-pregnancy complications; an introduction Int Jastd aids 1997;8:17-18

39. Ferris DG, Litaker MS, Woodward L, Heindrich J. Treatment of bacterial vaginosis: a comparison of oral metronidazole,

metronidazole vaginal gel and clindamycin vaginal cream. J Fam Pract 1995;41: 443-449

40. Greaves WL, Chungafung J, Morris, B Haile A, Townsend J L. Clindamycin versus metronidazole in the treatment of bacterial vaginosis. Obset Gynecol 1988;72:799-802

41. Hay PE, Morgan Dj, Ison CA et al. A longitudinal study of bacterial vaginosis during pregnancy, Br J Obstet Gynaecol 1994;101:1053.

PROFORMA

S.No.

Name :

Age :

IP No. :

SE Status :

Obstetric Code :

GA in weeks

LMP	
EDD	

DOA	
DO Del	
DO Dis	

Booked / Unbooked

Previous H/o Preterm Labour / Abortion

Admission - Delivery interval

General Examination :

Height	
Weight	
Temperature	

PR	
BP	
CVS	
RS	

P/A - Per abdomen examination

L/E - Local examination

S/E - Speculum examination

P/V - Vaginal examination

Investigations :

Routine :

Hb, Blood grouping typing

Urine albumin, sugar, deposits

HIV, VDRL

Specific Investigations :

Nature of vaginal discharge	
pH	
Amine Test	
Clue cells	
Gram stain Nugent's scoring Spiegel's criteria	

Mode of Delivery :

Neonatal outcome :

Birth weight :

Apgar 1 min :

5 min :

Neonatal complications :

Birth asphyxia

Respiratory distress syndrome

Meconium Aspiration Syndrome

Hyperbilirubinaemia

NICU admissions :

Neonatal death:

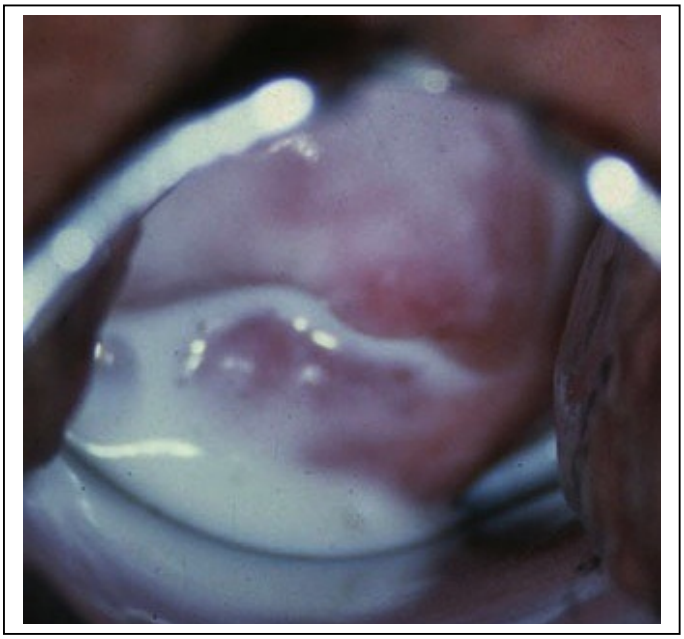
Maternal Outcome :

- Maternal temperature
- Uterine tenderness
- Nature of lochia
- Episiotomy wound infection

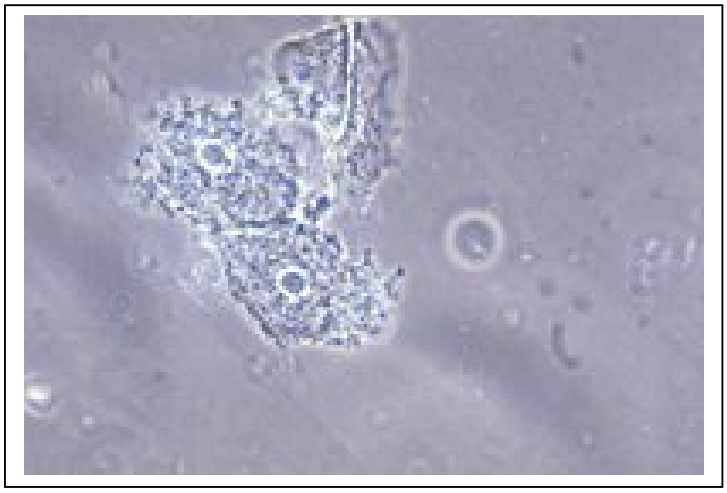
ABBREVIATIONS

APH	ANTEPARTUM HEMORRHAGE
BA	BIRTH ASPHYXIA
BV	BACTERIAL VAGINOSIS
DM	DIABETES MELLITUS
EWI	EPISIOTOMY WOUND INFECTION
GDM	GESTATIONAL DIABETES MELLITUS
H_2O_2	HYDROGEN PEROXIDE
LSCS	LOWER SEGMENT CAESAREAN SECTION
LN	LABOUR NATURAL
MSAF	MECONIUM STAINED AMNIOTIC FLUID
NPV	NEGATIVE PREDICTIVE VALUE
PID	PELVIC INFLAMMATORY DISEASE
PTB	PRETERM BIRTH
PTL	PRETERM LABOUR
PROM	PREMATURE RUPTURE OF MEMBRANES
PPV	POSITIVE PREDICTIVE VALUE
RDS	RESPIRATORY DISTRESS SYNDROME
STD	SEXUALLY TRANSMITTED DISEASE

NATURE OF DISCHARGE IN BACTERIAL VAGINOSIS



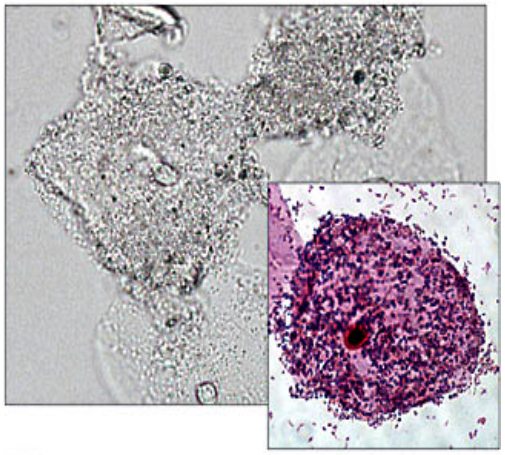
CLUE CELLS



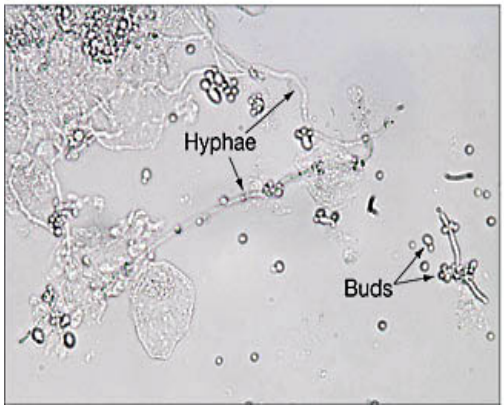
LACTOBACILLUS

A Normal Vaginal Epithelial Cells

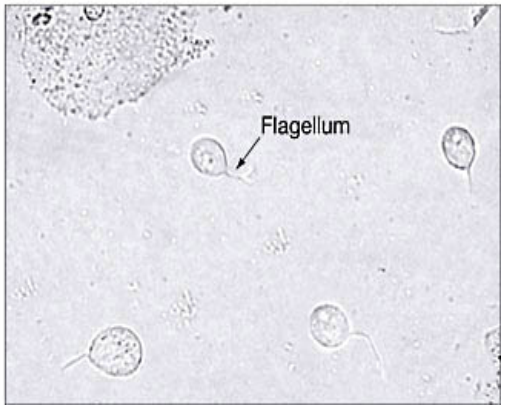
B Clue Cells With Coccobaccilli



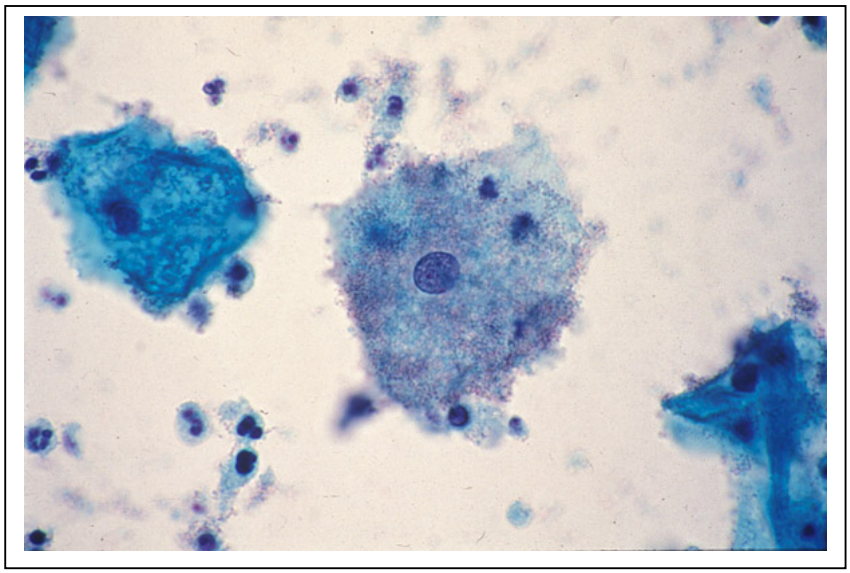
C *Candida*



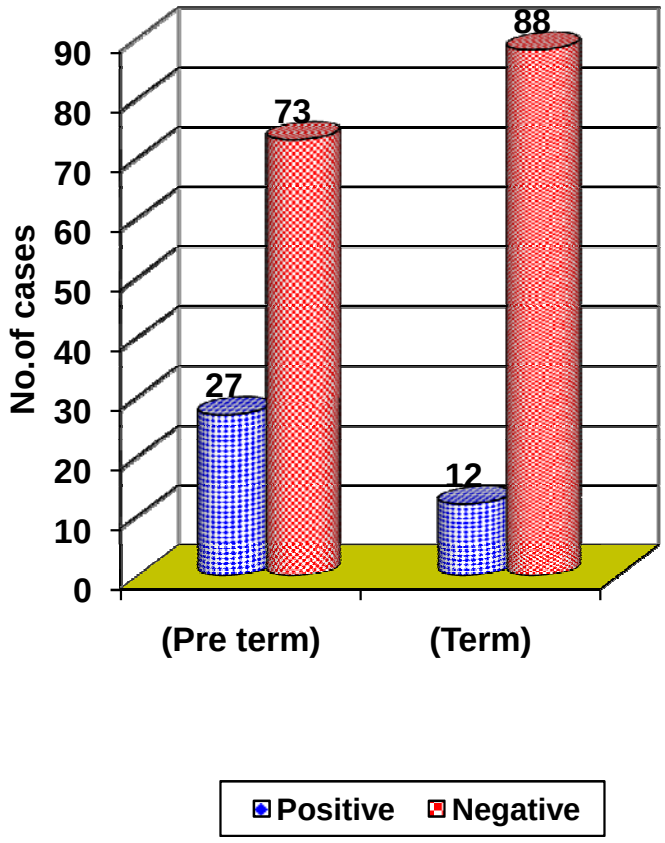
D Trichomonads



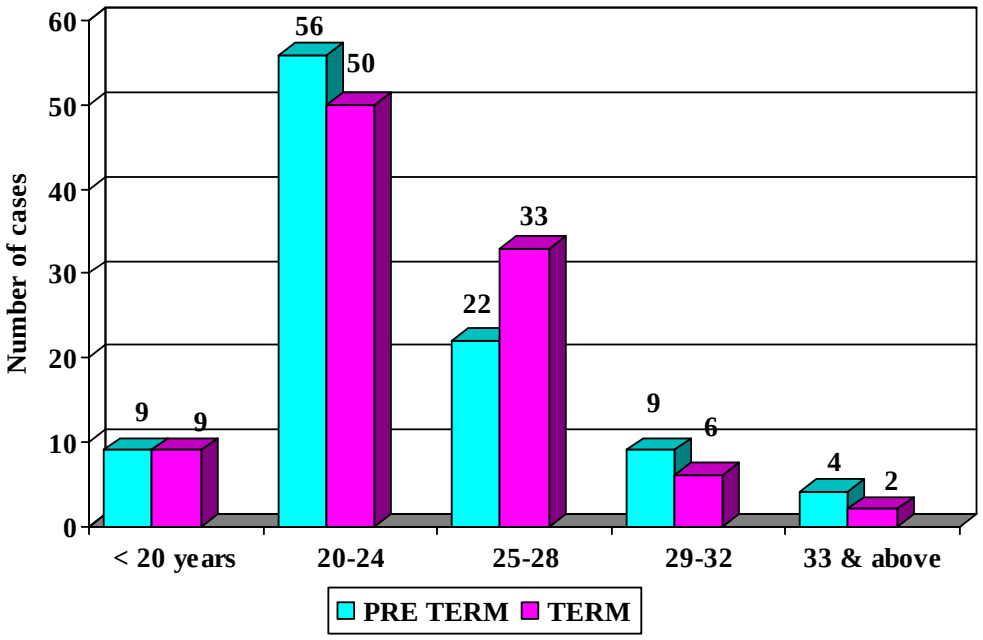
GARDNERELLA BACTERIA



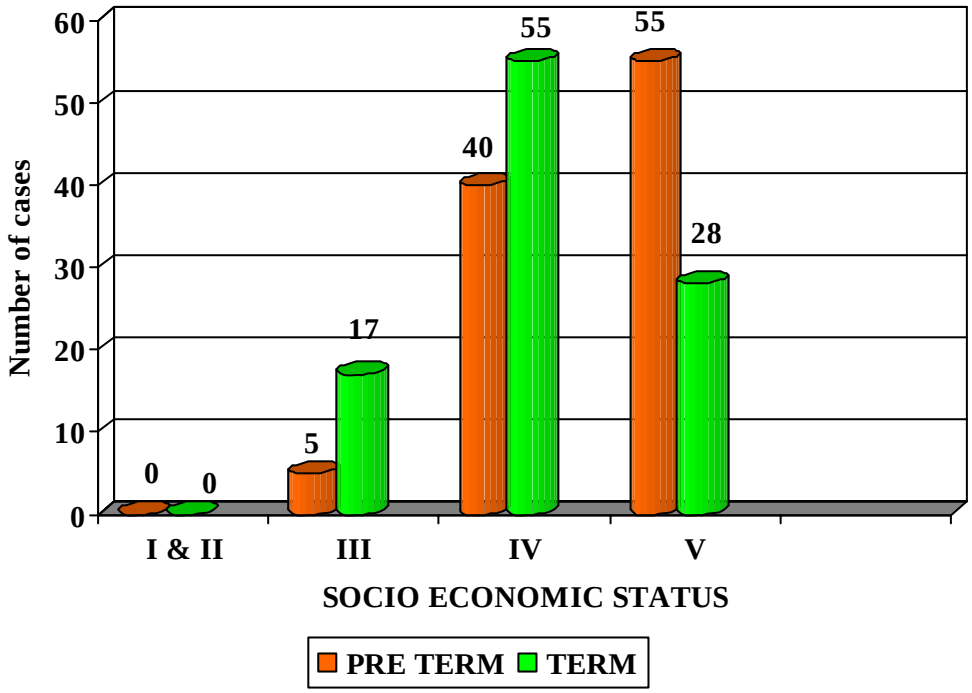
Distribution of Bacterial vaginosis among the subjects



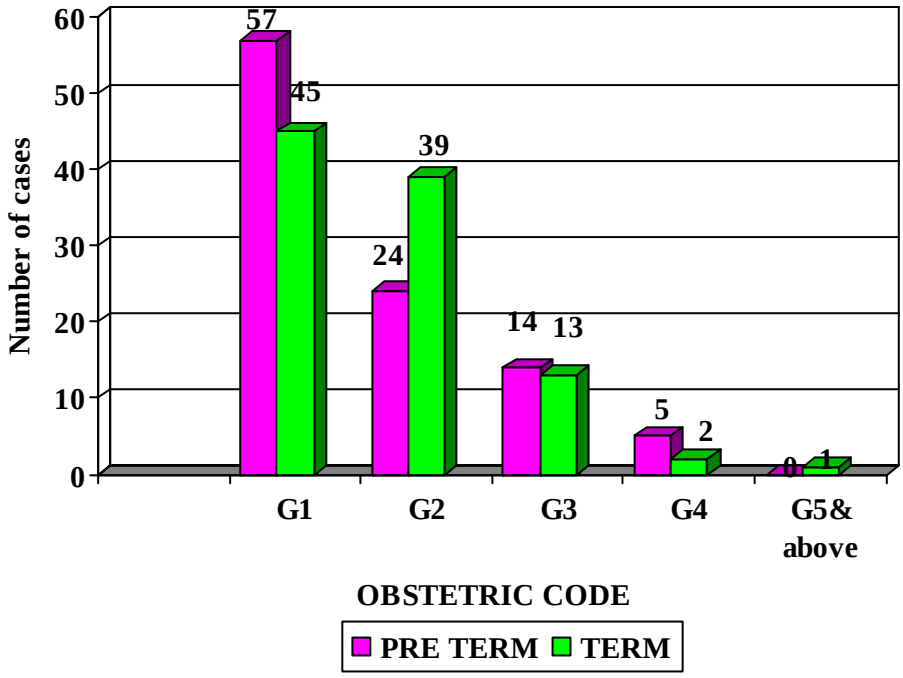
AGE DISTRIBUTION



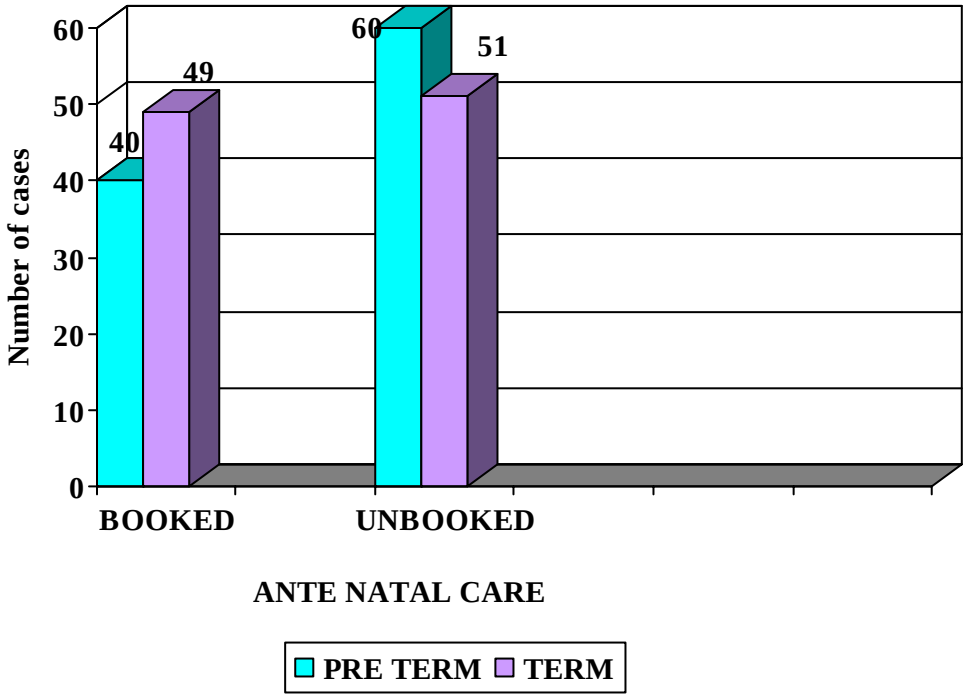
SOCIO ECONOMIC STATUS



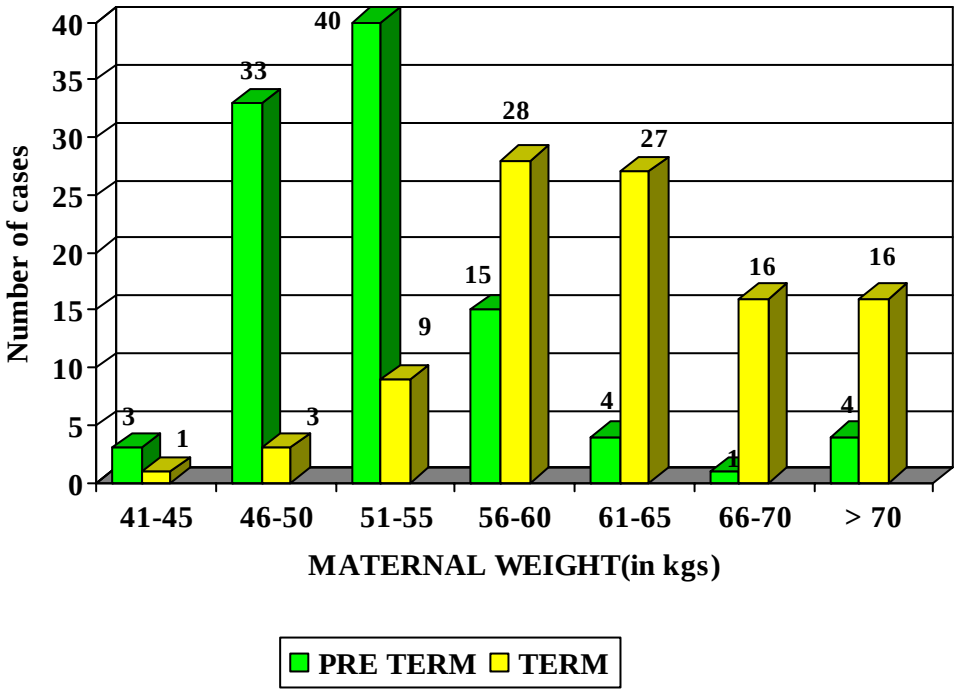
OBSTETRIC HISTORY



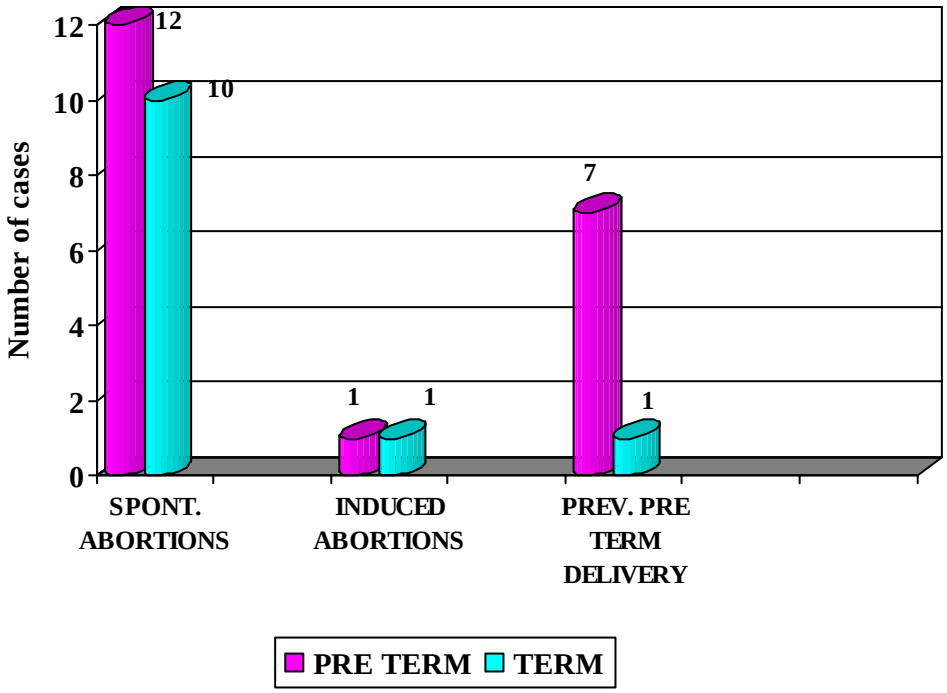
ANTE NATAL CARE



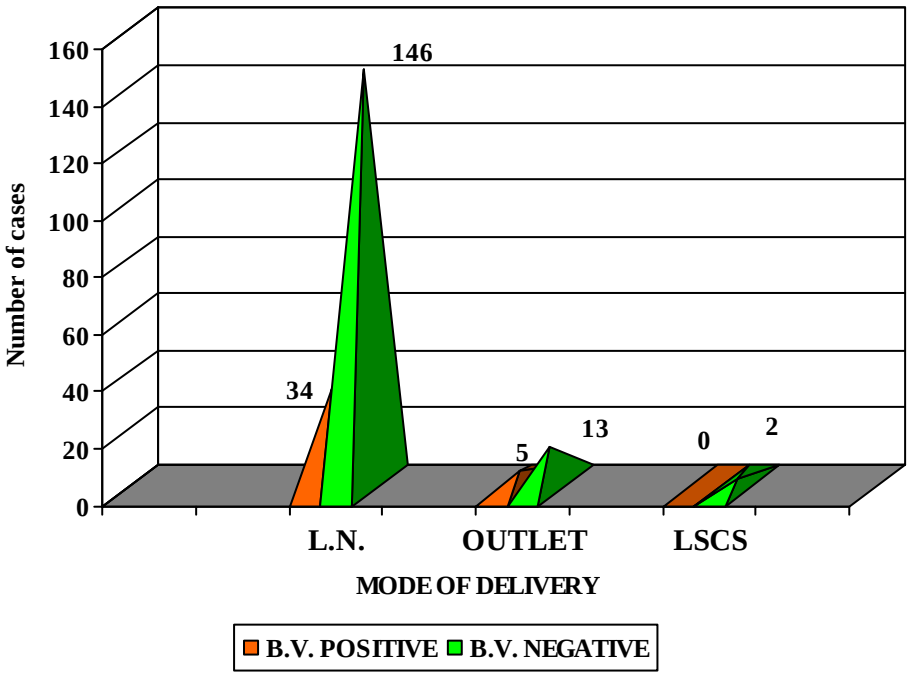
MATERNAL WEIGHT



PAST OBST. HISTORY



BACTERIAL VAGINOSIS & MODE OF DELIVERY



TERM CASES (CONTROL)

S.No	Name	Age	IP. No.	SES	Booked/Unbooked	Obs. Code	GA (WKS)	Previous H/O PTL/Abortion	Wt (Kg)	White homogenous Discharge	PH	Amine test	Clue Cells	Amsel's criteria	Gram stain		Mode of Delivery	Neonatal outcome				Maternal Complication
															Nugent's Scoring	Spigeis Criteria		B. W.	Neonatal Complications	Neonatal Admission	Neonatal Death	
1	Selvakumari	27	105580	III	Booked	Primi	38	-	78	-	4	-	-	-	1	-	LSCS	2.75	BA	+	-	-
2	Pattau Ilavarasi	22	106679	IV	Booked	G4P3L3	38	-	46	-	4.5	-	-	-	4	-	LN	2.8	-	-	-	-
3	Priya	21	106990	IV	Unbooked	G3A2	39	2 Spont. Abortion	53	-	5	-	-	-	0	-	LN	3.8	-	-	-	-
4	Lakshmi	24	107139	V	Unbooked	G2P1L1	38	-	58	+	6	+	-	+	7	+	LN	3.5	-	-	-	-
5	Priyadharshini	22	107307	IV	Booked	Primi	40	-	64	-	5	-	-	-	0	-	LN	3.8	BA+	+	-	-
6	Suganthi	25	44	V	Unbooked	G2P1L1	39	-	74	-	4	-	-	-	2	-	LN	2.75	-	-	-	-
7	Mala	34	918	IV	Unbooked	Primi	38	-	72	-	4	-	-	-	1	-	LN	2.7	-	-	-	-
8	Venkateswari	26	22033	IV	Unbooked	G2P1L1	40	-	64	+	5.5	+	+	+	8	+	Outlet	2.75	-	-	-	-
9	Janaki	24	22105	III	Unbooked	G3P2L2	39	-	68	-	4	-	-	-	3	-	LN	3.25	-	-	-	-
10	Muthulakshmi	25	23744	IV	Booked	Primi	39	-	68	-	4	-	-	-	3	-	LN	3.3	-	-	-	-
11	Jawahar nisha	36	23773	V	Booked	G6P4L3A1	38	1 Spont abortion	56	-	5	-	-	-	3	-	LN	3	-	-	-	-
12	sivaranjani	17	29368	V	Booked	Primi	39	-	64	-	4	-	-	-	1	-	LN	3	-	-	-	-
13	Deivanayaki	25	40469	V	Booked	G2P1L1	40	-	82	-	4	-	-	-	2	-	LN	3.6	-	-	-	-
14	Muthulakshmi	24	40487	IV	booked	G2P1L1	37	-	62	-	5	-	-	-	2	-	LN	2.5	-	-	-	-
15	Amsavalli	20	40679	IV	Booked	Primi	41	-	60	-	4	-	-	-	2	-	LN	3.2	-	-	-	-
16	Karthika	19	40683	III	Booked	Primi	39	-	64	-	4	-	-	-	1	-	LN	3.1	-	-	-	-
17	Pappathi	23	41393	V	Booked	Primi	40	-	67	+	5.5	+	+	+	8	+	LN	2.8	-	-	-	-
18	Mariyabeevi	23	55862	IV	Booked	G3P2L1	41	-	64	-	5	-	-	-	3	-	LN	2.9	-	-	-	-
19	Pandiselvi	21	58407	III	Unbooked	Primi	40	-	72	-	4	-	-	-	3	-	outlet	2.5	-	-	-	-
20	Vironikal	29	58548	V	Unbooked	Primi	38	-	82	+	6.5	+	+	+	9	+	outlet	2.7	BA+	+	-	-
21	Backiyam	21	61582	V	Booked	Primi	40	-	56	-	4	-	-	-	1	-	LSCS	3.75	-	-	-	-
22	Muthumari	19	66223	IV	Unbooked	Primi	40	-	72	-	4	-	-	-	2	-	LN	3	BA+	+	-	-

23	Sofia	24	67006	III	Unbooked	G2P1L1	39	-	70	-	4	-	-	-	2	-	LN	2.6	BA+	+	-	-
24	Pandiselvi	21	67077	IV	Unbooked	Primi	39	-	50	-	4	-	-	-	1	-	LN	2.75	-	-	-	-
25	Sivakavitha	19	67078	III	Unbooked	Primi	39	-	55	-	4	-	-	-	2	-	LN	2.8	-	-	-	-
26	Pandeeswari	26	67265	IV	Booked	G3P1L1A1	40	1 Spont abortion	64	-	4	-	-	-	0	-	LN	2.8	-	-	-	-
27	Jayakodi	28	67296	IV	Unbooked	G3P2L2	39	-	62	-	4	-	-	-	2	-	LN	2.5	-	-	-	-
28	Sara begum	25	68263	III	Booked	G2P1L1	39	-	58	-	4	-	-	-	3	-	LN	2.25	-	-	-	-
29	Hajeera	23	68278	V	Unbooked	Primi	40	-	61	-	4	-	-	-	1	-	LN	3	-	-	-	-
30	Karthigaiselvi	27	68547	IV	Unbooked	Primi	39	-	60	+	5.5	+	+	+	7	+	Outlet	2.8	-	-	-	-
31	Murugeswari	22	68950	V	Unbooked	G2P1L1	40	-	44	-	5.5	-	-	-	3	-	LN	2.6	-	-	-	-
32	Sathya	24	69011	IV	booked	G2P1L1	38	-	53	-	4	-	-	-	3	-	LN	2.4	-	-	-	-
33	Ramu	25	69020	IV	booked	G3P1L1A1	40	Abortion +	60	-	4	-	-	-	2	-	Outlet	3.1	-	-	-	atonic pph
34	Vanishree	20	69124	IV	booked	Primi	41	-	60	-	4	-	-	-	3	-	LN	2.5	-	-	-	-
35	Meena	23	69228	IV	Booked	G2P1L1	39	-	52	-	4	-	-	-	3	-	LN	2	-	-	-	-
36	Karpagam	25	69472	V	booked	G3P2L2	38	-	54	-	4	+	-	-	3	-	LN	3	-	-	-	-
37	Murugeswari	20	69900	IV	Unbooked	Primi	39	-	65	-	4	-	-	-	3	-	LN	2.75	-	-	-	-
38	Shanthi	22	69924	IV	Booked	G2P1L1	39	-	70	-	4	-	-	-	0	-	LN	3.1	-	-	-	-
39	Guruvammal	20	69975	IV	Booked	Primi	39	-	60	-	4	-	-	-	0	-	LN	2.6	-	-	-	-
40	Shanthi	22	69997	III	Booked	Primi	40	-	60	-	5	-	-	-	2	-	LN	3.5	-	-	-	-
41	Sivakami	20	70004	V	Unbooked	G2P1L1	39	-	58	-	4	-	-	-	1	-	LN	2.6	-	-	-	-
42	Shanmugasundari	26	70063	V	Unbooked	G2P1L1	39	-	60	-	4	-	-	-	4	-	LN	2.75	-	-	-	-
43	Tamilselvi	24	70113	IV	Booked	G2P1L1	38	-	59	-	4	-	-	-	3	-	LN	3.25	-	-	-	-
44	Murugeswari	20	70121	IV	Booked	Primi	38	-	58	+	6	+	+	+	7	+	LN	2.3	-	-	-	E W I +
45	Eswari	24	70402	V	Unbooked	Primi	40	-	65	-	4	-	-	-	6	-	LN	2.3	BA+	+	-	-
46	Murugeswari	23	70403	III	Unbooked	G2A1	40	1 Spont abortion	72	-	4	+	-	-	1	-	LN	3.1	-	-	-	-
47	Muthulakshmi	26	70459	IV	booked	G2P1L1	39	-	51	-	5	-	-	-	2	-	LN	2.6	-	-	-	-
48	Anitha	20	70585	IV	Booked	G2P1L0	38	-	58	+	6	-	+	+	7	+	LN	2.75	-	-	-	-
49	Dhanam	27	70685	IV	Unbooked	G4P1L1A2	38	2 Spont. Abortion	62	-	4	-	-	-	2	-	LN	2.75	-	-	-	Lumbar plexopathy
50	Thailyalnayaki	20	70763	V	Unbooked	G2A1	41	1 Spont Abortion	60	+	4	-	-	-	2	-	Outlet	3.5	-	-	-	-

51	Mahalakshmi	31	70888	IV	Unbooked	Primi	38	-	58	-	4	-	-	-	2	-	LN	2.25	-	-	-	-
52	Jayasudha	27	70912	IV	Booked	G3P2L2	40	-	64	-	4	-	-	-	2	-	LN	3.1	-	-	-	-
53	Ramuthai	20	70955	V	Bookekd	Primi	39	-	72	-	4	-	-	-	3	-	Outlet	3.5	-	-	-	-
54	Dhanalakshmi	27	71002	V	Unbooked	G2P1L1	40	-	66	+	6	+	+	+	7	+	Outlet	3.2	-	-	-	E W I + gaping +
55	Panchavarnam	21	71090	V	Booked	G2P1L1	38	-	58	+	6.5	+	-	+	7	+	LN	3	-	-	-	-
56	Pothumponnu	25	71404	IV	Unbooked	Primi	39	-	70	-	4	-	-	-	1	-	LN	2.7	-	-	-	-
57	Saraswathy	30	71406	IV	Booked	Primi	38	-	71	-	4	-	-	-	0	-	LN	2.75	-	-	-	-
58	Dhanalakshmi	29	71409	V	Unbooked	G3P2L2	39	-	56	-	5.5	-	-	-	2	-	LN	3.1	-	-	-	-
59	Pandeeswari	22	71644	V	Unbooked	Primi	39	-	61	-	4	-	-	-	1	-	LN	3.1	-	-	-	-
60	Azhagumoorthy	25	72211	V	Unbooked	Primi	41	-	65	-	5	-	-	-	3	-	Outlet	2.75	BA+	+	+	-
61	Kuthaladevi	23	72280	IV	Unbooked	G2P1L1	41	-	66	+	6	+	+	+	7	+	LN	2.8	-	-	-	-
62	Asinabanu	26	72299	IV	Unbooked	G2P1L0	39	PTL +	60	-	4	-	-	-	2	-	LN	2.6	-	-	-	-
63	Rajeswari	27	72475	IV	Unbooked	G2P1L1	41	-	62	-	4	-	-	-	3	-	LN	2.65	-	-	-	-
64	Saranya	19	72636	IV	Unbooked	Primi	40	-	55	-	5	-	-	-	3	-	LN	3.2	-	-	-	-
65	Selvi	22	72842	III	Booked	G2P1L0	40	-	70	-	4	-	-	-	3	-	LN	3.1	-	-	-	-
66	Kodimalar	25	72974	IV	Booked	G2A1	39	1Spont Abortion	56	-	4	-	-	-	3	-	Outlet	3.3	-	-	-	-
67	Selvalakshmi	19	41524	IV	Unbooked	Primi	40	-	64	-	4	-	-	-	2	-	LN	2.7	-	-	-	-
68	Kasthuri	22	41570	III	Unbooked	Primi	39	-	65	-	4	-	-	-	2	-	LN	2.6	-	-	-	-
69	Mathana	25	41585	V	Unbooked	G3P1L1A1	39	Abortion +	76	+	5.5	+	+	+	8	+	LN	2.75	-	-	-	-
70	Chellam	23	73305	IV	booked	Primi	39	-	60	-	5	-	-	-	2	-	LN	3.3	-	-	-	-
71	Mahadevi	23	73469	V	Unbooked	G2P1L1	39	-	58	+	4	-	-	-	2	-	LN	2.8	-	-	-	-
72	Ambika	25	73538	IV	Unbooked	G2P1L1	40	-	71	-	4	-	-	-	3	-	LN	3.5	-	-	-	-
73	Vijayarani	23	73604	III	Booked	G2P1L1	40	-	67	-	4	-	-	-	4	-	LN	2.8	-	-	-	-
74	Asanbanu	27	73609	III	Booked	G2P1L1	39	-	68	+	5.5	-	-	-	2	-	LN	2.75	-	-	-	-
75	Sarammal	28	73636	IV	Unbooked	G2P1L1	38	-	64	-	4	-	-	-	2	-	LN	2.25	-	-	-	-
76	Selvi	25	73841	V	Booked	G2P1L1	41	-	62	-	4	-	-	-	2	-	LN	2.6	-	-	-	-
77	Banupriya	22	73856	IV	Booked	Primi	39	-	62	-	4	-	-	-	3	-	LN	2.9	-	-	-	-
78	Pandeeswari	27	73863	III	Unbooked	G2A1	37	-	60	-	4	-	-	-	2	-	LN	2.6	-	-	-	-
79	Panchu	21	73881	IV	Unbooked	Primi	40	-	64	-	4	-	-	-	4	-	LN	2.6	-	-	-	-

80	Thilakam	27	41641	III	Unbooked	Primi	38	-	78	-	4	-	-	-	3	-	Outlet	3.25	-	-	-	-
81	Radha	22	41642	IV	Unbooked	Primi	39	-	58	-	4	-	-	-	1	-	LN	2.6	-	-	-	-
82	Thilagavathy	26	74145	IV	Booked	G2P1L1	40	-	62	-	4	-	-	-	2	-	LN	2.9	-	-	-	-
83	Mahalakshmi	24	74224	III	Booked	Primi	39	-	55	-	4	-	-	-	1	-	LN	2.6	-	-	-	-
84	Nagalakshmi	23	74444	IV	Unbooked	G2P1L1	38	-	62	-	4	-	-	-	1	-	LN	2.75	-	-	-	-
85	Sabeetha	30	74525	III	Unbooked	Primi	39	-	58	-	4	-	-	-	2	-	LN	3.2	-	-	-	-
86	Mahalakshmi	19	74675	IV	Booked	Primi	38	-	66	-	5	-	-	-	2	-	LN	3.2	-	-	-	-
87	Deivarani	24	74718	IV	Unbooked	G3P1L1A1	40	Abortion +	80	-	4	-	-	-	1	-	LN	3.8	-	-	-	-
88	Iswarya	18	74832	V	booked	Primi	38	-	56	-	5	-	-	-	3	-	LN	3.25	-	-	-	-
89	Sudha	19	74842	IV	Unbooked	Primi	38	-	68	-	4	-	-	-	5	-	LN	2.75	-	-	-	-
90	Mahalakshmi	31	75182	V	Unbooked	G2P1L1	39	-	72	-	4	-	-	-	1	-	LN	3.5	-	-	-	-
91	Muthurakku	21	75233	IV	Bookekd	Primi	39	-	68	-	4	-	-	-	3	-	LN	2.6	-	-	-	Fever +
92	Indira	23	75234	IV	Booked	G3P1L1A1	40	induced Abortion	78	-	4	-	-	-	2	-	LN	3.25	-	-	-	-
93	Fathimuthu Jebara	20	75248	IV	Booked	Primi	38	-	58	-	4	-	-	-	2	-	LN	2.7	-	-	-	-
94	Sangeetha	26	75258	IV	Booked	G2P1L1	41	-	54	-	4	-	-	-	3	-	LN	3	-	-	-	-
95	Velliyammal	25	75544	IV	Unbooked	G2P1L1	40	-	64	+	6.5	+	+	+	8	+	LN	2.75	-	-	-	-
96	Manimozhi	26	41719	IV	Booked	G3P2L2	39	-	49	-	4	-	-	-	2	-	LN	3.1	-	-	-	-
97	Kalaiselvi	27	76037	IV	Booked	G2P1L1	40	-	68	-	4	-	-	-	2	-	LN	3.3	-	-	-	-
98	Pitchiyammal	22	76124	V	Unbooked	Primi	41	-	62	-	4	-	-	-	1	-	LN	3.5	BA+	+	-	-
99	Brindhadevi	20	76135	V	Unbooked	Primi	39	-	64	-	4	-	-	-	1	-	LN	3	-	-	-	-
100	Alaguindira	22	76169	IV	Booked	G2P1L1	40	-	68	-	4	-	-	-	2	-	LN	3.25	-	-	-	-